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The Royal London

Ophthalmic Hospital Reports.

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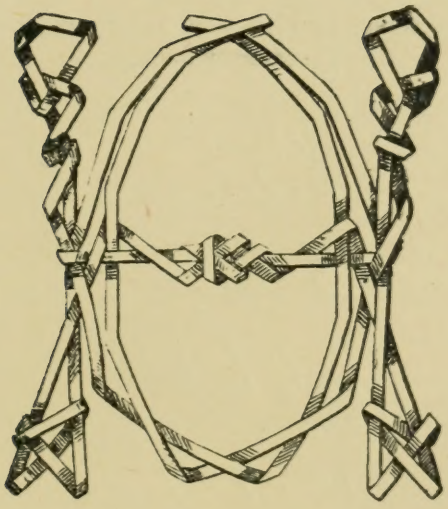

VOLUME FIFTEEN.

LONDON:

J. & A. CHURCHILL, 7, GREAT MARLBOROUGH STREET.

892263

LONDON:
HARRISON AND SONS, PRINTERS IN ORDINARY TO HIS MAJESTY,
ST. MARTIN'S LANE.

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The Royal London

Ophthalmic Hospital Reports.

Vol. XV. Part I. May, 1899.

RETROBULBAR OPTIC NEURITIS.

By E. Nettleship.

(A Clinical Lecture given at the Hospital on March 8, 1899.)

In the cases I propose to speak about the mischief is limited to one optic nerve;* the lesion affects the nerve behind the eye but in front of the brain, and it does not depend either upon changes in the eye itself or upon disease within the skull. The entire class, not a large one, is by no means homogeneous; the cases indeed form a rather mixed assemblage, and one of their most obvious common characters is that they cannot be classed anywhere else. The cases of symmetrical toxic amblyopia—with tobacco amblyopia as the type—are outside our subject.

The optic nerve, like other nerves, is subject to inflammation beginning in itself; whilst, on the other hand, disease may be communicated to the nerve by the various parts amongst which it passes between the chiasma and the eye—the orbital tissues, the optic canal, the sphenoidal sinus. It is therefore likely on pathological grounds that two chief families of the retrobulbar neuritis we are now considering exist; and we find in fact that an assignment of the cases to one or the other of the two pathological families just mentioned is usually justified on clinical grounds.

* Or attacks the other only after an interval.

We must, however, neither think nor speak dogmatically on this subject, for the opportunity of verifying the nature of any case of single retrobulbar neuritis must always be excessively rare—if indeed it should ever occur. And however sure we may feel that the pathological division into an *idiopathic* and a *symptomatic family* is correct, we shall continue to meet with some cases which, though undoubtedly belonging to one family or the other, cannot on clinical grounds, with our present knowledge, be confidently placed in either.

In the retrobulbar neuritis I am speaking of, usually, as already stated, only one optic nerve suffers, and if both should be attacked there is an interval commonly of months, sometimes of years, between the two.

In Case 134* the interval between the first eye and the second was three months; in Case 115, six months; Case 132, about nine months; Case 93, 12 months; Case 116, five years; Case 28, several years. An interval of days only is very infrequent (Case 74).

The failure of sight is generally rapid, the climax being generally reached in from one day to four or five; but it is never really "sudden" as in embolism or arterial thrombosis. The degree of failure of vision varies extremely in the idiopathic cases, from the faintest "mistiness" of objects in the centre of the field to total loss of perception; it bears no uniform relation to the early ophthalmoscopical changes; and recovery of good sight is possible even in the worst cases, provided that the improvement sets in tolerably soon. In the symptomatic family slight forms are much less common and the prospect of recovery not nearly so good.

* The numbers refer to 148 cases, being all of which I have notes, consisting of 123 which were arranged for the discussion on Retrobulbar Neuritis at the Ophthalmological Society (Trans., vol. xvii, 1897), and 25 that have occurred since. Some of the earlier ones were published in a paper in the Transactions of the same Society in 1884 (vol. iv). Of the whole series (148) 28 are omitted as incomplete, or doubtful, and the lecture is therefore based on 120 cases.

The field of vision is invaded in various degrees, but in cases of every degree and kind the central part is involved; and in the slighter varieties this is as a rule the only part affected. The defect is often more or less fan-shaped, spreading towards the periphery though not constantly in the same direction.

Something can often be learnt, particularly in the milder cases, from the patient's description of the defect of sight.

A lady of 34 (Case 63) could see 6/9 with the affected eye, but the letters were "misty" and there was "a dark mark near the centre." Another lady of 35 (Case 66) said that the defect had come on during one day in the form of a "spot" and of "a darkening" of the objects she looked at; when I saw her two weeks later V. was 6/6, but there was still "a patch of mist," and I found a relative scotoma (for red) a little up and in from the centre; a month later colours and light looked alike to the two eyes.

A lady of 29 (Case 68) said that when the sight of her right eye failed a white surface "looked like mottled soap," and that print and coloured objects had the appearance of being "gapped" (interrupted). When seen 10 days after the failure, V. of the affected (R.) eye was 6/18, with much loss of temporal part of field and no ophthalmoscopical changes; pupil not noted; in six weeks V. was 6/9; in five months 6/9, still with no definite changes at O. D.

The direct reflex action of the pupil is lowered, even in the milder cases, more than the loss of visual acuteness seems to warrant, and in the bad cases it is of course almost or entirely lost.

Pain about the affected eye is a very common symptom, though, as in so many other maladies, its value in diagnosis requires adjustment for each patient. It is of two sorts: spontaneous pain behind the eye and in the corresponding temporal district; and pain caused or increased by movements of the eye—sometimes by movement in one particular direction—or produced by pressing the eyeball back into the orbit. Observe carefully that the pain, however bad, is

limited to the side of the affected optic nerve, and is not accompanied by vomiting. In the idiopathic cases the pain usually precedes the loss of sight and ceases when, or soon after, this occurs; it is a less frequent feature in the symptomatic cases, but when present in them is often more severe and much more prolonged, continuing for days or even weeks after the sight has failed.

I have never seen and very seldom heard of any external signs of inflammation, and such slight symptoms of it as are occasionally mentioned by the patient may perhaps be accounted for by his having kept the eye covered or closed during the painful stage.

A young man (Case 26) spoke of the eye having been red during the stage of pain that preceded the failure of sight. Another patient (Case 27) said that the lids had been rather swollen; and a third (Case 52), also a young man, spoke of the eye having been a little blood-shot during the severe pain of the early period.

Some ophthalmoscopical changes* are usually present almost from the first in the idiopathic cases, but they are often very slight and occasionally quite wanting; such early changes—various degrees of haziness of the disc, sometimes with decided swelling, but without hæmorrhages and with only moderate venous engorgement—point to a true, if not as a rule very gross, acute inflammation of the nerve. In some of the worst symptomatic cases, however, no change whatever can be seen at the disc for some weeks after the loss of sight.

Later on in cases of both families the disc almost always becomes pale, often very pale, whether the sight improves or not. In very few is it true that the nerve passes through an

* P.S.—I see that Dr. Sym, in a recent paper on retrobulbar neuritis (American Journ. Med. Sci., February, 1899), states (p. 154) that ophthalmoscopical changes are "usually absent in the early stages and may be absent altogether." I think that a careful comparison of the two discs will show slight but decided differences within a few days of the failure of sight in a large proportion of cases.

attack causing, even for a time, severe loss of sight, without some appearances either of early inflammation or late atrophy of the disc.

The inconstancy in the degree of visible papillitis and in the date and degree of the consecutive pallor doubtless indicate differences not only in the severity of the morbid process, but also in the distance at which it is seated from the eyeball.

In Case 9 (F., æt. 36) the disc was becoming pale two weeks after the onset.

In Case 45 (F., 38) slight haze and swelling of O. D. with V. 6/12 partly, one week after onset: O. D. appeared quite normal and V. 6/6 four weeks after onset.

Case 72 (M., 17) showed congestion and slight haze with V. = shadows at ninth day. O. D. pale and V. 6/12 at end of fourth week.

Case 72 (F., 27) four days after onset, papillitis with swelling = 3 D. and V. = fingers at 3 feet; seven weeks, O. D. pale all over, V. 6/6.

In Case 78 (F., 28), seventh day, haze, but no swelling; four weeks decided pallor.

Case 97 (F., 30) three days slight haze and swelling, V. = fingers eccentrically; four weeks clearer and getting pale V. 6/12.

In Case 82 (F., 29) one week, O. D. slight hazy, V. 6/18, with a large scotoma from centre up and out; six weeks O. D. pale all over and clear.

Cases 68 and 122 showed no decided changes from beginning to end; the former has been already quoted, the latter (122) was as follows:—

Rapid severe failure of one eye with very slight changes at the time or afterwards. Complete recovery in a month. No personal complications, but family history of nervous disease, and optic nerve disease in a brother. Condition five years later.

Mrs. G., æt. 38 (Case 122), was sent by Dr. Hague on January 3, 1884, for recent blindness of the L. eye; she could see my hand in the temporal part of the F., but nowhere else, pupil motionless

to direct light. The only changes were slight haze of O. D., and a decided whiteness of the coats of several of the arteries on and a little beyond the O. D.; all the arteries pulsated easily on pressure (*i.e.*, no indications of arterial blocking); colour of O. D. normal.

She said that as she was at needlework about half past 11 A.M., on December 28 (five days), "a feeling came as if there were something across the sight of the left eye," by 5 P.M. the same day she could not see the V, VI, and VII on the clock face, and by 7 P.M. she "could not see the clock face at all, though she could still see the fire."

Dr. Hague reported that there were no signs of renal or other organic disease. She had often had "rheumatism" in knees, wrists, and shoulders since she was 15, but never rheumatic fever; had always been subject to bad megrim attacks, consisting of headache on the L. side with "forcing" pain in the eye, followed by pain at back of neck and profuse micturition, sometimes with nausea, but no vomiting; the visual spectra of megrim had never been observed.

Treatment.—Iodide and grey powder; blisters. January 10 (12 days from failure of sight, six from beginning of treatment). L. counts fingers everywhere, except at centre and nasal parts of F. Retinal arteries as before, veins decidedly too large; there is now some diffuse haze of retina; vitreous n. Commencing salivation.

17th (three weeks from failure) L. 20/70, and reads 8 J., "the word itself is plain, but around it there is a sort of purple mist. At night things look pale, and people's faces look ghastly." On trial with Bull's coloured spots there was no true colour-blindness, but confusion between red and yellow, green and blue; but I did not examine the F. for insular defects. R. V. 20/30.

January 26 (four weeks) L. 20/30 and 2 J. The fundus "I should say now shows nothing unnatural"; no choroidal disease. R. 20/20 and 1 J., some large patches of exposed sclerotic below the O. D., probably an incomplete congenital insular coloboma: no disease elsewhere.

In March, 1889 (*five years later*), Mrs. G. came to St. Thomas's Hospital, thinking her eyes had failed again, but Mr. Laws found that she only needed better glasses, V. being full in each eye, and

that the fundus of the L. was (as at my own last note) normal in appearance, and F. (measured on perimeter) full.

Mrs. G.'s mother died at 53 of a "fit," having been subject to "hysterical fits," which "turned into epilepsy" since she was 40; she was not paralysed.

Mrs. G. was one of eight children, all living except one, a brother (Thomas H.) who had been under my care at St. Thomas's Hospital in 1881 for failure of both eyes, which I had diagnosed as due to tobacco, though his friends thought it was due to chlorodyne. The fact was that he was an inveterate smoker and drinker of chlorodyne. Some time after he had left me he was in an asylum for two years, where he was kept from chlorodyne but was allowed to smoke. Soon after coming out he resumed his old habits, became utterly demoralised, and died in 1884 nearly blind.

The diagnosis of retrobulbar neuritis is seldom difficult if attention be paid to the cardinal points, viz., the mode and rapidity of failure of vision; pain; the pupil; the acuteness of V. (refraction being, of course, correct or corrected); the field examined on the perimeter, but specially the central area tested for insular defect by means of coloured (red or green) spots of various sizes; the ophthalmoscopical appearances.

Retrobulbar neuritis of exceptionally rapid onset and high degree is occasionally difficult to distinguish from thrombosis of a branch of the retinal artery or from possible hæmorrhage into the sheath, until we find on careful inquiry that the sight was lost rapidly but not suddenly. It is important to distinguish the condition from congenital amblyopia of one eye, whether associated with a squint or not. Still more important is it not to confuse retrobulbar neuritis with hysterical amblyopia, an error into which we may be led by the frequency of this neuritis in neurotic women: the distinguishing marks are found in the state of the pupil and field, in the mode of onset and in the pain when present, apart from whatever the ophthalmoscope may reveal. Whilst maintaining that the disc usually shows some

inflammatory changes early in the case, I freely admit that such appearances may be absent or equivocal; and I therefore regard the investigation of the symptoms and the state of the pupil as more important than the ophthalmoscopical examination in distinguishing an early stage of retrobulbar neuritis from hysterical blindness of one eye. Another difference is in the severity of the loss of the sight; hysterical amblyopia is, I think, never slight in degree, whereas the retrobulbar disease may be of any degree from the very slightest to total blindness. In retrobulbar neuritis vision is not bettered by any assistance or suggestion; in many of the functional ("hysterical") cases the patient, though unable to see at first, improves after staring at the test types, whether the near or far set, and finally reads each successive smaller size with a jerk or a rush, especially if coaxed by false lenses, which are represented as gradually increasing in "strength."* Central scotoma is, I believe, very uncommon in hysterical cases, whilst, as already stated, it forms a constant feature in the retrobulbar disease.

The only instance in which I have seen "hysterical" amblyopia take the form of central loss of field was the following:—

Relapsing functional central Amblyopia of one eye in a boy whose other eye was defective from Central Choroiditis.

Master W. (P. 19—45) was 13 when his father first brought him to me on September 19, 1889, for recent blindness of the L. eye. The boy said that just two weeks ago at half-past ten in the morning, something came over the middle of the sight of the L. eye, got to its worst during that day and remained the same till I saw him on September 19 as above.

The condition then was:—

R. V. less than 6/60; a patch of pigmented choroidal atrophy with fibrous deposit just at Y. S. and some other patches rather further further out; O. D. clear and rather paler than L. Direct

* I mean a + and − lens of equal value superposed, beginning with + and −0·5 D. and going on to + and −1 D., &c.

reflex action of P. less good than L. All the fundus changes appeared to be old, and he had been told by an ophthalmic surgeon several months before, that this eye had probably been useless for some time.

L. V. less than 6/60; with − 0·25 D. = 6/60; not 20 J. Rough examination of F. shows a large central blind area with good periphery. P. quite normal to light, acting better than the R. O. D. of better colour and less clear than R., and retina shows watered silk reflex. My note was "probably the R. O. D. is the morbid one." The defect appears to him as a bright red cloud with a fleecy border in the sunlight, but as a yellow cloud in ordinary light. The F. measured next day, showed a large rudely circular scotoma, absolute at the centre; at the periphery of the scotoma, which about corresponded with the 30° circle, the test dot moved centripetally becomes dim, and then about 5° further in it "goes into a yellow mist" and entirely disappears. Periphery of F. to white (10 mm. spot) full and prompt.

Knee jerks very brisk.

On October 4, the field was in all respects the same and V. no better; but the P. was still quite normal to light; and the O. D. showed no decided change. In spite of the activity of the pupil I thought, in view of the disease in the other eye, that treatment for retrobulbar neuritis was advisable, and I asked Dr. Maurice Duke, who sent the patient to me, to keep him in bed and give small doses of mercury until, if possible, the gums were touched; and to use several small blisters. He took mercury four times a day for three weeks with no marked physiological effect, but at about this date (Wednesday, October 30th), the boy said "the cloud had got very thin," and on 31st, he could see well. On November 8, I found he could see 6/6 partly and 1 J. fluently, O. D. n. as before.

The previous history was to the effect that there had been some complaint of the sight the previous Christmas, for which it was thought best for him to stay away from school for a term. Then in July he had an attack of blindness of L. eye stated to be like the one above described, but only lasting a few days; and again in August he had a similar attack which came whilst he was watching his float in fishing, and got well in a few days.

He was brought again in February, 1890, as blind as ever with

the L. (the fourth attack), unable to count fingers at 6 inches, but with the P. and O. D. normal; the "cloud had grown over the eye" during one day a month previously. I did not again measure the field, but put him to bed in St. Thomas's Hospital, and made a show of treatment by the inunction of simple vaseline; he was soon well.

He was an intelligent quiet good boy, not apparently neurotic. First of all I suspected malingering, then hysteria, then after taking F. I veered towards retrobulbar neuritis for a time though there was nothing positive in favour of it. The loss of sight was of course functional in this boy's L. eye, and I suppose it took the form of central scotoma because the other eye had previously lost its central vision from organic disease.

Central localised choroido-retinitis often causes a well-defined insular defect in the field, and when the mischief comes on rapidly the diagnosis from retrobulbar neuritis may be difficult.

The distinction between an idiopathic and a symptomatic case of retro-bulbar neuritis is sometimes quite easy; in other instances, as has been indicated, it may be difficult or impossible. To recapitulate the main differences: in the symptomatic family pain, when it occurs, is often worse and more lasting than in the idiopathic cases, visible changes at the disc sometimes much delayed, and recovery of sight less hopeful; paralysis of other cranial nerves (from the first to the seventh) and signs of disease of bone at the base are fairly common in this family, whilst indications of chronic disease of brain or spinal cord—often incipient only—are far more frequent in the idiopathic family. But it must be pointed out again that the members of what I call for convenience the symptomatic family, vary much among themselves, far more than those classed as idiopathic, and it is to the idiopathic form that I ask most attention to-day.

You will see from the table* that the idiopathic family is much the larger, and for that reason, and even more because this form of disease of the optic nerve, whether lastingly

* A tabular classification drawn up for the lecture.

single or eventually double, is apt to prove a harbinger of some form of spinal sclerosis, this family is the more important form of the two. The idiopathic series or family can, without going into needless minutiæ, be subdivided into (A) a single, and (B) a double, group, and as will appear later on, the group of double cases is again divisible. The single cases (Group A) are by far the more numerous, even allowing that some of them are incorrectly classed and eventually become double. Thus in 88 cases in family I (idiopathic form), Group A (only one optic nerve affected) contains 73, Group B (both optic nerves attacked) in all its varieties, only 15.

The prognosis for sight in the whole idiopathic family is, speaking generally, good. The slighter cases often recover vision that is, for all ordinary purposes, perfect; and even in the worst examples improvement from nothing, or mere hand movement, to 6/12, more or less, is not uncommon. The final result is usually reached in from five to ten weeks from the date of failure. The following are typical single idiopathic cases:—

Rapid failure of L. to fingers at 3 feet, with scotoma and definite papillitis. Rapid improvement to almost perfect recovery with pale disc and some residual light-sense defect, in five to six weeks.

(Notes by Dr. F. A. Hayward.)

Ellen B., æt. 27, came to Moorfields on January 27, 1897. (Case 77 O. P. letter.)

She said that on 20th, when at a theatre, she noticed pain about the L. eye when looking sideways. The pain continued for about four days, but does not seem to have been severe. On 23rd she found L. eye going, and it rapidly became worse, but as atropine was used by her doctor a day or two later, the exact progress of the failure cannot be stated.

On 27th (four days after failure of V.) the L. eye could only count fingers at 3 feet; pupil dilated (from atropine). O. D. swollen 2 or 3 D.; hazy and streaky; no hæmorrhages, but veins

rather distended; refraction at Y. S. about H. 2 D., at O. D. 5 D. R. V. 6/6; H. estimated at O. D. 2 D. F. showed no peripheral loss, but a horizontally oval scotoma, from centre to blind spot, was easily found with a 10 mm. white test object.

Dr. Hayward's careful examination of the organs and the nervous system revealed nothing unnatural, and the family and personal history were negative, except a liability to sick headaches. Such a sick headache with vomiting occurred the day before she noticed the orbital pain.

Treatment.—Calomel purge: iodides and a blister 1 inch square every alternate night (three blisters in a week).

February 3 (10 or 11 days from failure, one week after admission). V. of L. is much better, 6/36 or 6/24. Appearance of O. D. as before.

February 24 (three and a half weeks). L. 6/9, but only 6/24 of Bjerrum's gray types (R. seeing 6/9 of same gray types).

March 17 (six and a half weeks). L. 6/6, 6/12 of Bjerrum's types (R. = 6/9 of Bjerrum's types). L. O. D. quite clear, uniformly pale all over, and not now at all swollen. No scotoma could now be found on the most careful trial.

Severe acute case, with slight but definite changes passing into pallor of disc. Almost perfect recovery. No field charts.

Sister K., æt. 30 (Case 79, P. 40—62), apparently in excellent health, and not overworked, or living in any way an unnatural or unhappy life, has worn glasses for H. since she was 13, and for some months past. + 2·5 D. cyl. with + 1 D. sph. for each eye ordered by Mr. Attlee. The R. eye has never been so good as the L.

She had always been liable to asthma, but this had been much better since she had left London for a country sisterhood, six months before this narrative begins; she had been somewhat constipated when she first entered the sisterhood.

It was for failure of the L. (or working) eye that she came to Moorfields, on October 24, 1896, stating that the sight had failed rapidly three days before, and that five or six days before the affection of sight there had been some pain in the L. brow and in the eye, especially when looking to one side. The pain had

already diminished before she came. Mr. Holmes Spicer found *on admission* that V. in L. eye was reduced to counting fingers in upper part of F., and was worst at centre of F. The colour of a red card 4 inches square could be recognised only in the upper part of the F. She described a strained feeling on looking to her R., but the movements were full, and there was no tenderness on pressing the eye through the eyelid. Eserine had been used before she came, and the reaction to light could therefore not be satisfactorily observed. The ophthalmoscope showed a "slight ill defined haze and swelling of the disc as compared with the R."

She was ordered to take 6 (and later 8) grains of iodide of potassium, and 10 of salicylate of sodium thrice daily.

On November 8 (two weeks after failure) she said V. was considerably better. The O. D. was paler, but still rather hazy. Direct action of p. present, but much less than that of right eye. Treatment continued.

On November 19 (four weeks). L. with her glasses 6/12 or 6/9, but only 6/36 of Bjerrum's "A" types; disc clearing and getting paler. This compared with R. (with her glasses) 6/12, and Bjerrum's "A" 6/18, or 6/12.

Thus the V. of L. for black and white had improved in the most striking manner, though the sense of light contrast, as shown by the ability to read gray types, was much behind that of the healthy, but originally somewhat defective, R. eye.

On December 10 (seven weeks). L. 6/6 just, but Bj.'s "A" only 6/18 slowly though correctly (against R. Bj.'s "A" 6/12 partly).

May 3, 1897 (six months). Objects still look "lighter" coloured to the L. than to R. eye. V. L., with a slight change in her glasses 6/9, not so well as R., Bj.'s "A" 6/18.

R., with a slight change in her glasses, 6/9 partly, Bj.'s "A" 6/12.

Thus, whilst the form sense of the affected eye recovered almost fully, the light contrast sense did not recover in quite the same proportion.

In this case Dr. Emerson reported that the organs and the functions were apparently healthy. We did not examine the knee reflexes. The patient's parents had been double first cousins;

the mother died 16 years previously, of rheumatic fever; the father was living, and subject to eczema, which was said to be gouty.

March, 1899. Condition of eyes as at last note, 6/9 with each eye with the best correction, the L. (neuritic) eye not quite so well as R.; Bj.'s. types 6/12 with each.

pid failure of left to almost total loss of perception: definite papillitis. Quick recovery of V. and subsidence of papillitis.

(Notes by Dr. F. A. HAYWARD.)

Kate F., æt. 19 (Case 126 O. P. letter), came to Moorfields on March 31, 1897, stating (1) that six weeks previously her L. eye had ached and become rather dim, but had got well in a few days; (2) that six days ago the same eye had failed again, and become blind between then and the date of coming to the hospital: this time there had been no aching or pain either in the head or about the eye. She had been exposed to cold wind when cleaning windows the day before the second failure.

On admission, L. has only p. l., cannot distinguish fingers against the light; pupil contracts a very little to the full light of the electric ophthalmoscopic lamp, but the contraction is not maintained. Some tenderness on pressing the eye back into the orbit, but no pain from movements of the eye. O. D. redder than the R., hazy, especially below, and decidedly swollen (2 D), veins rather large, but no hæmorrhages; no other changes; refraction at Y. S. Em. R. 6/6, Em., O. D. n., and much clearer than L.

No nasal symptoms. Patient seems healthy. Catamenia regular: was "anæmic" two years ago, but is not so now; no history of rheumatism. Her mother suffers from rheumatic gout, affecting the foot chiefly.

Treatment.—A blister 1 inch square to the temple every night for three nights: iodide of potassium and salicylate of sodium in moderate doses.

April 3 (10 days from failure), p. l. is decidedly better, but still requires the contrast to be strong.

April 10 (17 days). V. better, but not 6/60. Swelling of O. D. now only 1 D.

April 17 (three weeks). V. of affected eye 6/36.

April 24 (four weeks). V. of affected eye 6/12; O. D. now seen best without any + lens. No later note. No note of F. or of knee reflex.

Acute severe case. Good recovery. Notes not exhaustive.

Miss B., æt. 19 (Case 110, P. 20, 205) was sent by Dr. Watters of Stonehouse. She had always been short-sighted, and the vision of L. seems never to have been so good as the R.

On the evening of Friday, May 23, 1890, she noticed some mistiness of vision, and the next morning found the R. (or better) eye nearly blind.

When she came on June 6 (two weeks) V. of R. was fingers at 4′; the direct reflex action of p. was very little; the disc hazy, congested, and slightly swollen, no hæmorrhages, vessels normal. L. 6/24 improved to 6/18 by − 2 D. cyl. with − 1 D. sph., but result indefinite both at this and subsequent trials. The shadow test and estimation by ophthalmoscope showed M. 3 D. in vertical meridian, 1 D. horizontal.

Ordered to take iodide and mercury, to have blisters to the temple, and wear dark glasses.

On July 7 (six weeks) R. with correction 6/18.

On October 3 (five months) R. "as good as the L." she says. Careful estimation showed M. 3·5 D. vertical, and with this correction she had 6/18, not more. There are no notes of F. or colours, nor of final ophthalmoscopical appearances; and the recovery seems to have been only partial. But the general features of the case are characteristic and well marked.

A few days before the eye failed she had been travelling by railway with her face to the engine, and the window open; but she did not feel cold. The patient had never had arthritic symptoms; but her mother and mother's mother were subject to gout, and there was also gout on the father's side.

A case in which, with very little loss of V. for black and white, the patient complained of failing sight in one eye, and great loss of light-difference sense was found, together with scotoma; marked pallor of O. D.

(Notes by Mr. H. PARKER.)

Thomas J. D., æt. 21 (Case 129, O. P. letter), compositor, came to Moorfields on 2nd of October, 1897, complaining that for a week past he had not seen properly with his R. eye. The eye had been aching a good deal, and he had some toothache lately, but there had been no pain or tenderness about the eye, and no headache.

Except that the knee jerks were found to be very brisk, there was nothing to be noted; he was pale (indoor occupation), did not smoke, and denied ever having had any venereal disease; family history gave no assistance.

Though he said he saw badly with the R. eye, Mr. Parker found V. in this eye 6/9 raised to 6/5 in + 0·5 D., the L. V. being 6/6 with and without + 1·5 D.

At this stage it would have been easy to dismiss the case as indefinite, for the disc appears to have shown no decided alteration. But the pupil was observed to be rather larger than the other (*i.e.*, less active to direct light), and on then trying V. with Bjerrum's types, Mr. Parker found a very great difference between the light sense in the two eyes, the R. with correction seeing only 6/36, whilst the L. saw 6/9. There was a small scotoma just above the centre of the F. in the R.; no peripheral loss.

October 16 (two weeks later, or three weeks after failure began) the above observation was repeated with the same result, and there was now decided pallor of R. O. D. as compared with L.; no change whatever at the Y. S.

On December 18 (nearly three months) V. of R. seemed not quite so well, viz., with + 1·5 D. 6/12 Sn. and 6/60 Bj. against 6/6 of both types in L. with the same lens. The R. O. D. is noted as showing much pallor. But six weeks later (January 29, 1898, four months or more) R. again sees 6/9 Sn with + 1·5, and after this he did not come.

The case was slight from beginning to end, and apparently

slower in coming to an end than some cases are. It might easily have been overlooked had not the patient been accustomed by his trade to accurate observation, and had Mr. Parker not quickly appreciated the leading features, viz., the state of the pupil, the very marked lowering of the light sense and the gap in the field.

A similar slight case. Notes not so complete.

Mrs. D., æt. 45 (Case 88; P. 30, 157), was sent by Dr. Morison on May 4, 1894, for a defect of two weeks' duration in L. eye with a "bruised feeling at the top when the eye is moved." Is rheumatic, and has "barometric joints."

May 4, 1894. R. – 4·5 D., 6/9 well; Bj.'s "A" = 6/9.

L. – 4·5 D., 6/9 "blurry"; reads word by word, and has to look sideways to it. Bj.'s "A" = 6/24 barely. Reflex action of p. less than in R. Relative scotoma at centre of F. and above; periphery not measured. O. D. blurred; edge invisible by direct examination; no swelling.

Blisters and salicine.

Smoked glasses.

June 14. R.—4·5, 6/6 partly; Bj.'s "A" 5/6 partly.

L.—4·5, 6/6 partly, but not so well as R.; Bj.'s "A" 6/12. No ophthalmoscopic note.

Not seen again.

The following was probably a case of slight retrobulbar neuritis, but the micropsia and the retinal haze point rather to central retinitis of low degree. It at least illustrates the occasional difficulty in diagnosis already alluded to.

Mrs. Walter B., æt. 69 (Case 105; P. 22, 61), sent by Dr. Schacht on January 26, 1891, for slight defect of R. found out accidentally three weeks before; a little mist as if with dust on it, and type looks only half the size it looks to the other eye; all things look a shade darker than to the other eye; a picture with this eye looks as if in the shade; with the L. it looks as if in sunshine.

Now, R. p. and T. n. (p. rather > L.); Hm. 4 D.; V. 6/6 partly

L. Hm. 3 D.; 6/6 well.

R. (after mydriasis) O. D. and surrounding retina slight haze and decidedly different from L.; Y. S. quite n.

A well marked relative scotoma for 5 and 10 mm. red and for pale pink from fixation point outwards "exactly as in tobacco." Periphery of F. (to hand) full.

(R., no trace of scotoma).

March 16. R. w. + 4 D., 6/6 partly, and still looks "smaller" than to L.; Bj.'s "A" 6/18; O. D. certainly a little paler than L., especially on Y. S. side. Scotoma to 5 mm. pink still present, but not > 5° or 10° each way from centre.

L. w. + 3 D., 6/6 well; Bj.'s "A" 6/9.

Of the double cases (idiopathic family Group B) it seems that we may for the present distinguish B 1,—Cases which resemble those of Group A (Single Cases), but suffer one or more subsequent attacks like the first, and affecting either the same eye or alternating between the two; and which are complicated by loss of power or loss of sensation in one or more limbs (usually the legs), that may (like the loss of sight) be temporary, but often settles into something permanent. Repeated attacks of this optic neuritis seem usually to leave some degree of permanent defect, but not necessarily a high degree.

There is no doubt that some of the Single Cases (A) are of the same essential nature as the double ones just mentioned, and may sometimes, if observed long enough, be seen to pass into the latter. At present I have notes of only some six cases of the Group B 1, and select the following:—

Miss —— (Case 93; P. 29, 251). Early in 1894, she being aged 29, the L. eye failed, and when I saw her early in March, V. was 6/60 and 15 J., the O. D. pale with some diminution in size of the arteries, and the direct reflex action of p. much less than in the R. The F. does not appear to have been examined. 12 months later (April, 1895) the L. had improved to 6/18; the O. D. was pale as before, and F., though of full expanse, showed an absolute scotoma close to the centre. The R. (hitherto good) had now been failing for a fortnight, and its V. was 6/36, with

slight haze of O. D., a small absolute scotoma but full expanse of F. Knee jerks now very brisk, but no ankle clonus.

June, 1895. R. and L. each see about 4/60 and 16 J., both O. Ds. pale; ps. act to light.

November. Thinks sight worse, and says that it varies a good deal and often; R. (2nd eye) 3/60; L. (1st eye) 6/24; Fs. about as last time. Lately some pain and weakness in L. leg "as if it would give way."

April, 1896. Reports "eyes better on the whole, but still vary; left much better, but right still very dim."

February, 1899. Her brother, a medical man, reports that about three years ago her sight again got much worse for a time but improved again, and that now, with both eyes, she can read some of 14 J., and do a little sewing, play chess, &c., but cannot see 6/60; that she cannot walk far, and has decided weakness of L. hand, arm, and leg.

Miss —— (Case 115; P. 16, 134), æt. 21, was first seen June 21, 1888, for failure of sight in the left eye which had begun four or five weeks before, and got worse for the first few days. As she had quite lately gone through a severe distress, and had several years previously more than once behaved strangely and been considered morbid and fanciful, the eye failure was thought to be hysterical both by her doctor and parents. Some numbness of the legs up to the waist which had come on when the eye failed seemed likely to be of the same nature.

June 21, 1888. Well nourished and apparently strong. Is reticent, but though professing to make light of her ailments, is really very pessimistic about her sight. Sleeps badly and reads late into the night.

With L. eye barely sees 6/60; F. shows large, dense scotoma for red; O. D. decidedly pale, especially on Y. S. side; vessels n.; no appearances of embolism; P. acts less freely than R. Decided lowering of sensation to pin-pricks in both legs; more in R. than L.

Ordered blisters to temple, and to wear smoked glasses; and sent for opinion to Dr. (now Sir William) Gowers as I could not agree that the case was hysterical. Dr. Gowers thought that the

leg symptoms might be due to a temporary affection of the cells of the posterior columns of the spinal cord, and were thus not ordinary hysteria; but that the legs would probably recover, and that practically therefore the symptoms might be called functional.

July 3. L. sight still only 6/60; F. shows no contraction; loss of perception of green in all parts; red is correctly perceived, but with hesitation, and worst close to centre of F.; blue promptly seen in all parts of F. Action of pupil to light markedly less than in other eye. O. D. as before; considerably paler than R., especially on Y. S. side, and also a degree less transparent than R., and vessel's coats rather thicker.

To take iodide and lithia and mercury (gr. 1 of tannate), and have mustard to spine.

July 27. L. has improved (from 6/60 barely) to 6/24 and 6/18 after intent looking, and can read in the hand from 10 J. (fairly) up to 2 J. (words). Glasses do not help.

September 7. L. now sees 6/12, and some of 6/9. On November 1, Dr. Gowers, who had seen her again, found a tendency to stumble when going up steps, and a little want of equilibrium with the eyes shut and feet together, and thought the development of permanent organic disease more likely.

December 11. L. sees 6/9 slowly, but well. For a week past, R. has been failing, with pain in or behind the globe. Now can but just see hand moving in the upper outer quadrant of F. P. almost motionless to light, and becomes very large when the L. is covered. O. D. red, rather hazy, and veins too full. Has of her own accord resumed iodide and mercury. To use blisters again.

December 21. R. now sees hand easily in all parts of F., except at centre and at down-in quadrant. O. D. is slightly swollen (H. 2 D. as compared with H. 0·5 D. at Y. S.).

January 22, 1889. R. sees 3/60, but not quite 6/60. O. D. still hazy, but temporal side getting pale.

February 26. R. O. D. much clearer, and Y. S. side paler. Now sees 6/60.

Was not seen again until May 17, 1889, when she came saying she was worse, and on trial, V. was very bad in both eyes. (This fresh failure was certainly recent in L. The R. might have remained unaltered since February; but I think it had

improved and failed again, for had it remained at 6/60 ever since February, the very marked improvement which now soon took place would have been unlikely to occur.)

Now (May 17) R. barely sees 6/60, F. of full size, but shows a dense scotoma from centre outwards.

L. 6/36, a similar scotoma from centre outwards but not so large as in R. F. of full size. Ps. act to light. O. Ds. about as last time. Confuses all red, green, and gray colours.

No further note until June 11, 1891 (two years), when R. sees 6/12 and L. 6/9 partly, and easily reads small print. Both O. Ds. very pale on Y. S. side, but no other changes.

The next note is July 22, 1892, when she said she had lately had a short attack of double vision "caused by the left eye"; this had now quite passed off. V. was the same as last time, not quite 6/9 with the two eyes together.

In the summer, 1893, again had double sight, chiefly when looking to her left, probably L. external rectus.

November 1, 1893 (five years after first note). Now no recognised diplopia. Print sometimes gets confused or swimming both with and without her weak + glasses; also distant objects looked at fixedly, either with both eyes or either eye singly become "unsteady," "much like rain running down a window pane." Perhaps this may be due to minute nystagmus made worse by effort. Complains a good deal of pain at back of neck (about seventh vertebra). V. same, 6/9 partly with both together The L. leg still drags somewhat, and is weak, with effort she can manage to put the foot upon the chair seat. Knee jerks (now tried by me for the first time) very brisk.

In April, 1897, the L. eye again failed rapidly with some days of pain in turning the eye about. I saw her on 12th with V. only 6/60 in this eye eccentrically (central scotoma passing also upwards from centre), P. acts less to light than R. O. Ds. as before.

In August, V. was 6/9 as before the attack. Sir William Gowers saw her again about this time and told me she had undoubtedly a sclerotic lesion of the cord, probably focal myelitis in the dorsal region.

One of this lady's elder brothers is also the subject of a form of spinal sclerosis with central blindness of one eye from partial

optic atrophy, the other eye failing slowly many years later. I have seen him at intervals from 1890.

The chief events and dates in his case were :—

1879, æt. 19. Double vision with drooping of eyelid (he does not remember which eye). Recovered in a few months without treatment.

1884. Failure of R. eye during one day with some pain, could not see his own hand with the eye. Had much advice and was thought by some to be malingering, because there was so little sign of disease in the eye. Sight improved a little but never became useful, and when I saw him in 1890 (six years after failure) at æt. 29, he could just read 16 J. with the eye by looking sideways, and there was a large loss in the F. from the centre up and out. O. D. atrophied. L. 6/6 F. full (perimeter) and no scotoma; doubtful pallor of outer side of O. D. Ps. act to light and accommodation, but R. less active to light than L. Articulation slow and catching; he walks carefully as if not at ease on his legs; under treatment for "general weakness." Denies ever having had intercourse.

In August, 1892, the eye condition was the same, and the L. continued to see 6/6 well, though its O. D. was somewhat pale. But he was getting much more ataxic and weak, articulation was more difficult, and his mental power seemed less than before.

In May, 1898, he came again, and this time the L. had sunk to 6/18 barely and 8 J. The R. still read 16 J. Ps. acted somewhat to light. Internal recti acted very imperfectly and R. diverged; no ptosis; other ocular movements full; rapid nystagmus of both. O. Ds. as before, L. much less pale than R., central vessels about normal. Incontinence of urine; constipation; speech jerking, gait jerking and weak; needs two sticks, and a servant to help him.

Several attacks of loss of sight, followed by recovery but leaving the discs pale. Various other temporary paralyses of motion and sensation. Patient under observation nearly twenty years.

Miss W. (Case 123, P. 7—161) gave me the following account :—

At the age of about 16, in 1876, after much family trouble

(money losses) she had an illness, accompanied by headache, loss of sleep and some "cramps" in limbs, during which she became nearly blind of both eyes. The late Mr. James Adams told her at Moorfields, she would soon get well, and she did, vision recovering "perfectly in both eyes" in six weeks.

Remained well, acting as a teacher, till 1880, when, at æt. 19, she again had "numbness and cramps," and the R. eye (not both eyes) again went rapidly almost blind. The eye improved and the other symptoms passed, and for about 18 months she was as usual.

During nearly the whole of 1882 she was under the care of a colleague for repeated failure of vision, alternating between the two eyes, but (as the Hospital letter showed) never very bad in both eyes at the same time. In August of this year, she came to me wishing another opinion. She was then an energetic, emotional, but very intelligent person of good frame and well nourished; no tremors, but some "numbness of the arms" (patient's statement). Complained of seeing colours badly, and of difficulty in judging the distance of objects because they looked too small. R. 6/60 and 14 J.; O. D. very pale all over, and arteries too small; P. large and sluggish to light. L. 6/12 and 1 J., O. D. pale especially on outer side, but much less so than R., and arteries not diminished; P. sluggish to light. Fs. to white full; marked confusion of red and green wools with R., and the same but less marked with L. in which, though no scotoma could be defined, colour vision was best eccentrically. Refraction Em. in both. No defect of smell nor dropping of fluid from the nostrils.

A year later (October, 1883), better in all ways and V. better (R. 6/18; L. 6/6 slowly), but said objects still looked small and far away to the R., and that long gazing either at far or near objects caused dizziness of R. and brought on headache.

On June 9, 1886 (aet. about 26) the L. (better eye) failed again rapidly and badly, but had partly recovered (to 6/12) when she came to me on the 26th. This time she also had double vision due to paresis of R. external rectus, and again suffered from headache and retching. R. 6/18 as before. She took small doses of iodide for about three months, improved in health and lost the diplopia.

On May. 10, 1890, after two or three weeks of pain first as general headache then localised in or behind the eyes, L. failed again, the pain ceasing when the sight went. On 19th, I found V. of L. only fingers at 12 inches, and direct reflex action of P. bad (worse than R.) O. D. as before, and showing no inflammatory appearance. R. as before, 6 18. V. of L. gradually improved again and by the end of July was 6 9, but became dim after a short effort. In this attack she again took iodide, and mercury was tried but caused diarrhœa.

In the late spring of 1893 after nursing her mother through a pulmonary illness, Miss W. was ill again (it was called influenza) and in June her L. leg became "numbed and felt as if tied round with a tight cord" and lost power, so that she had difficulty in going up and down stairs; it got better but then her R. arm followed in the same way so that she lost power of writing legibly, could not hold a cup and had to have her food cut up—the eyes did not suffer this time. I saw her in November, when the leg and arm had recovered, but the leg (L.) still "dragged" when she was tired and she considered her hand-writing (R. hand) still unsteady. Knee jerks extremely brisk. There were no noticeable tremors or nystagmus, but she had an anxious agitated manner and was much thinner. The discs had not changed in appearance since I first saw her, the R. being still paler than L., and V. was unaltered (R. 6 18, L. 6 9 partly); some qualitative loss in the nasal part of L. F., and to a less degree in the temporal part of R. I have not seen her again, but in 1895 she wrote, in very depressed spirits, that in spite of the recent illness and death of her mother, sight was keeping the same. She would then be about 35.

The remainder of Group B of the idiopathic family (comprising nine cases) contains some six examples of a different form which, for present convenience, I have called the Harding cases, after the name of the patient in the first case of the sort that I saw. The mark of these cases is rapid failure of one eye, shortly followed by similar failure of the other, and by quick and good recovery of both without recurrence of blindness or any nerve complications.

Case 74.—Male, æt. 23, 1883 (published as Case 11 in Trans.

Ophth. Soc., vol. iv, p. 203). R. had one or more fleeting attacks of dimness, and then failed rapidly to less than 6/60 in two or three days with much loss of F., involving centre and part of the periphery; complete recovery to 6/6 in three weeks, with some pallor of O. D. L. began to fail a week after R., went through a similar attack, with loss of F. of same character, but different in detail, and recovered perfectly in two to three weeks, also with pale O. D. Pain over the brow and pain in ocular movements preceded the failure of each eye.

Except that he was anæmic, and had lost nearly all his molars of both upper and lower sets, there was nothing to be made out as to possible cause. He had never smoked, and had been absolutely continent. Seen again 1894 (11 years) with V. 6/6 in each eye, and O. Ds. as before; in good health.

In the above case both O. Ds. showed a little haze with decided pallor, especially on the outer side, within six days of the failure of sight.

Mrs. —— æt. 30 (Case 7, Moorfields O. P. letter, 1889). Rapid failure of L.; two weeks later fingers at 2 feet, marked papillitis (swelling 2 D.); direct reflex of P. bad. Six weeks from failure 6/12; three months 6/6 partly; Bj. 6/18; four years 6/6, O. D. pale and clear.

R. failed a month after L., and in a week V. = only hand movement; papillitis as in L.; two months from onset 6/12; Bj. 6/36; four years 6/9, O. D. pale. No nerve symptoms.

A few days before the first (L.) eye failed, she had a blow by a wooden handle on the L. brow, and this was followed by a good deal of pain behind the eye. No evidence of syphilis could be obtained.

We may now attempt some conclusions as to the meaning of idiopathic inflammation of one optic nerve, or of both nerves with an interval; the seat and nature of the local disease, the prognosis, and treatment.

Sex.—Of the 88 idiopathic cases 58 were in females, only 30 in males—two females to one male.

Age.—Of 86 (out of 88) cases in which the age when the attack occurred is given (or in double and complicated cases,

the age at which the disease began), 69 were only 17 being 41 or more. Further, of the years limit, 60 were between 20 and 40; below puberty (taken as 15), and of these t much below that age.

If we now take the distribution of the to age, we find amongst the 69 cases bel limit 48 females to 21 males, or $2\frac{1}{4}$ to 1; patients above 40, the males numbered 8, practical equality.

These figures show conclusively that th neuritis is especially a disease of the most period of life, and that during that period than twice as subject to it as men.*

Apart from age and sex, we can in a ce the cases discover something in the patie may fairly be credited with a share, predispo in the attack, but in many nothing is as ordinary enquiry.

Exposure to cold after the depression or simply after or during physical fatigue, is nine of my cases, and this number is no mark.

A very definite history of gout in near in about six of the series, and knowing the which such an enquiry is often made, we must too as much less than the whole truth. giving this history were all seen in private, a

more often than any other shortly before the optic ne
and it appears to be answerable for some very s
attacks.

There was a history of typhoid fever just before th
attack in one or two, and of some form of malaria in t
three others; four cases in all.

Syphilis was certain in two of the idiopathic serie
probable in three others; but in the two cases where i
certain, the primary disease had occurred respectively 1
20 years before the eye attack, and in one of these the p
gave a clear history of malarious fever a few weeks
his eye failed. I think therefore we may, for the pr
conclude that syphilis is very seldom the cause of idio
retro-bulbar neuritis, except when it causes a gumma
upon the optic nerve; and such cases, of which I bel
have seen several, I purposely exclude, placing them
symptomatic family. Very different is the share tak
syphilis, whether recent or remote, in causing by one me
another a symptomatic retrobulbar neuritis. In my sy
matic family I have provisionally placed 32 cases, and sy
was certain, or almost certain, in about 17 of these—a
one-half—the primary disease dating from a few mon
many years prior to the optic neuritis.

Lastly, the history shows in a considerable number o
of idiopathic retrobulbar neuritis a predisposition to n
functional disturbance or organic disease of the ne
system. The excessive frequency already pointed out
optic nerve disease in women at and below middle
only one indication of this. Not only are many

actual disease of the nervous system in connection with quite 27 of the 88 cases—nearly 1 in 3. Of the 27, 7 were male and 20 female.

Slowly progressive double optic atrophy with loss of knee jerks, or the latter with loss of the pupillary light reflex, arouse a strong suspicion of incipient tabes dorsalis (locomotor ataxy). A very large proportion of these cases occur in men, and there is a history of syphilis in a considerable number.

An attack of acute retrobulbar optic neuritis usually single, or if double with much delayed symmetry, generally ending in partial or complete recovery should, I think, always cause some anxiety as to the future onset of disseminated sclerosis or some other disease of the cord, not tabes. The great majority of these cases occur in women during the sexually active years, scarcely one-third being in men. Syphilis appears to take little, if any, share in causing the attack.

Optic atrophy in the early stages of tabes often means, I believe, that the limb phenomena will be delayed or perhaps never fully developed. Will it, perhaps, be found in the future that disseminated sclerosis beginning as single retrobulbar neuritis shows less than its usual tendency to become generalised? However this may be if only one optic nerve be attacked, there is evidently less chance of escape from the general symptoms in cases where the second optic nerve becomes affected; for in no less than 6* of my 15 double cases the patient was more or less paralysed.

It is difficult, with our present imperfect knowledge, to understand the occurrence of acute disease in a portion, apparently well-defined, of only one optic nerve, the active process lasting for a certain fairly constant period, and then passing off.

The rapidity of onset points to either inflammation or slight infiltrating hæmorrhage, and of the latter,—hæmor-

* Cases 28, 76, 93, 111, 115, 123.

rhage from a minute intrinsic vessel in the optic nerve,—we have, I think, no clinical knowledge (the phenomena of bleeding into the sheath-space are different). The character of the pain points to the orbital part of the optic nerve as the seat of the inflammation and the frequency of early—however slight—inflammatory appearances at the disc can best be explained by a lesion near to the eye. The almost constant involvement of the central, or para-central part of the field indicates disease of those nerve fibres that come from the yellow-spot region and that lie on the temporal side of the nerve near the eyeball and in its axis further back. The scotoma often spreads out in a fan shape from the centre of the field towards or up to some portion of the circumference,—not the same sector of the field in all cases;* this seems to show that the morbid process tends to affect a wedge-shaped portion of the nerve, the apex of the wedge being at the macular fibres, the base at some (not always the same) part of the periphery.

If we suppose, with Mr. Marcus Gunn, that the arterial twig supplied by the central artery to the macular fibres in some way dominates the special liability of these fibres, we cannot, it would seem, make the same vessel responsible for the varying wedge of disease. And yet if we accept as probable that some (perhaps not always the same) morbid state of the blood furnishes the foundation of the attack, the a-symmetry of the disease would be explicable only by supposing that the poison acted on the nerve fibres indirectly, inducing changes in the walls, or inflammatory obstruction in the channel of certain minute blood-vessels or lymphatics in the nerve. Speculation apart we need not refuse to consider the fundamentally toxic origin of retrobulbar neuritis simply because of its a-symmetry; for gout is often a-symmetrical in single attacks, and this not only when perhaps

* The direction of the sector of loss in the field is, according to my observations, usually outwards (up-out, down-out, or horizontal); but in a certain number the base of the sector has been inwards and in a few directly upwards.

located by injury or pressure, but in the iris and sclerotic where no such influences have occurred; again herpes zoster though almost always one-sided, is sometimes caused by arsenic taken internally.

In the treatment of retrobulbar neuritis, when seen early, I believe in the usefulness of counter-irritation and the administration of iodides and often of mercury. But we know too little as yet of the natural history of the disease, to speak strongly about the efficacy of any particular treatment, and allowance must be made, in the present state of knowledge, for apparent differences in the cause. Nevertheless it is true that most of the severe cases that have made a good recovery in my hands have done so under vigorous treatment.

A NOTE ON THE ELASTIC TISSUES OF THE EYEBALL, AS SHOWN BY SECTIONS STAINED WITH ACID ORCEIN.

By E. Treacher Collins,

Assistant Surgeon to the Hospital.

The following description of the elastic tissue of the human eye is based upon the examination of a series of sections stained with acid orcein.

This method of staining has been introduced and elaborated by Unna, for demonstrating the elastic tissue in the skin (Maratsb. f. prakt. Dermatol, vol. xix, 1894, p. 397; Zeit. f. Wiss. Mik., vol. xii, 2, 1895, p. 240; Lee's Microtomist's Vade Mecum, 4th ed., p. 451).

In sections of the eye the tissues of which it is composed are of such varying density that they have to be treated in different ways for the elastic tissue to be shown in all parts. The length of time which suffices for staining elastic fibres in a soft tissue like the skin is not sufficient to show them in such tough tissue as the cornea and sclerotic. The length of time which is most suitable for the sclerotic and cornea is too long for the uveal tract. If the elastic fibres of the former are well shown, the latter will be much overstained, and if the elastic tissue in the latter is just right, that in the former is hardly picked out at all.

It does not, therefore, seem possible by this method to get one section of the eye showing the elastic tissue in all parts well stained.

In the cornea, Descemet's membrane stains much deeper than any other part, and in sections which have been allowed to soak for some considerable time (24 hours), it can be made to stand out a reddish-brown colour, the substantia propria remaining a dull pink. (Pl. A, Fig. 2.)

Bowman's membrane stains precisely the same colour as the substantia propria, so that this method of staining adds additional evidence to that which has been previously

brought forward as to the difference in character between Bowman's and Descemet's membranes, and as to the similarity in character of Bowman's membrane and the substantia propria.

It further emphasises the inappropriateness of the term anterior elastic lamina, as applied to Bowman's membrane.

The fibres of the ligamentum pectinatum, into which Descemet's membrane divides up, stain deeply with the acid orcein like Descemet's membrane itself. Its outermost fibres which pass into the sclerotic can be traced a long distance backwards, further than would be expected from other methods of staining, some way beyond the termination of the space of Fontana. As they proceed backwards they expand, so as to extend ultimately through about half the

Diagrammatic section of an eye. The elastic tissue staining with acid orcein being marked in red.

width of the sclerotic, having between them bundles of white fibrous tissue, equatorially and meridionally arranged.

This method of staining picks out in the sclerotic a number of delicate elastic fibres, finer than those usually met with in the skin. In sections these are seen cut in various directions, those cut transversely appearing as little dark red dots. Those cut longitudinally for the most part run parallel with the bundles of white fibrous tissue, and have a wavy course.

The outer layers of the sclerotic contain more elastic fibres than the inner, and they are more numerous posteriorly than anteriorly. Around the entrance of the optic nerve there is quite a dense plexus of them.

The lamina cribrosa is largely composed of elastic fibres, which extend across the nerve fibres from the plexus in the sclerotic on each side. From the lamina cribrosa a delicate network of elastic fibres extends backwards, around the central blood vessels of the optic nerve.

In both the pial and dural sheaths of the optic nerve there are numerous elastic fibres. In the former they are like those in the sclerotic, fine and delicate, but run a more tortuous course. In the dural sheath the elastic fibres are thicker than those in the sclerotic, but have the same wavy character, and run parallel to the bundles of white fibrous tissue.

In the tendons of the recti muscles where they are inserted into the sclerotic, thick elastic fibres are deeply stained by the acid orcein, they are especially numerous in the sheath around the muscles.

This arrangement of elastic fibres in the sclerotic which I have described, corresponds, I find, very closely with what was demonstrated by Professor Satler, at the 25th Ophthalmological Congress, at Heidelberg, in 1896. He employed Spaltenholz's method of staining, which shows up the elastic fibres black against a yellow background.

In the ocular conjunctiva many stout elastic fibres are seen lying in the tissue, between the epithelium and the sclerotic.

The elastic tissue in the inner coats of the blood vessels is picked out by the acid orcein in all parts of the eye. In the iris this elastic membrane in the blood vessels is the only elastic tissue shown.

In the choroid and ciliary body the membrane of Bruch stains deeply. I have also found the nodules of hyaline substance so often met with in pathological specimens on the inner surface of this membrane, stain in the same way.

The capsule of the lens, like Descemet's membrane, can be made to stand out a reddish-brown colour, while the lens fibres, like the substantia propria of the cornea, remain a dull pink. (Plate A, Fig. 1.)

The fibres of the suspensory ligament also stain deeper with the acid orcein than the surrounding tissue.

PLATE A.

FIG. 1.—A portion of the crystalline lens and its capsule, showing the latter deeply stained with acid orcein.

FIG. 2.—A section of the cornea, showing Descemet's membrane deeply stained with acid orcein. Bowman's membrane remaining the same colour as the substantia propria.

REPORT ON AN EYE HAVING SOME UNUSUAL CONGENITAL PECULIARITIES.

By C. Devereux Marshall, Curator.

The subject of this report was a man, William M., aged 32, who was first seen in 1887 with extensive detachment of the left retina, colobomata of both irides, and a large coloboma of the right choroid which could be seen with the ophthalmoscope, while the choroid on the left side was not visible.

He attended again in June, 1898, having for the previous five months suffered a good deal of pain in the left eye, which, as it was blind and appeared to be affecting the right eye, was excised. Patient had always been short-sighted, and the left eye was never so strong as the right, the vision of which with Sph. −11 D., Cyl. −4 D., Ax. Vert. 6/24. The left eye had been quite blind for about 12 years.

The eye was removed and hardened in formol, and the following condition was noted :—

The cornea is hazy, the iris discoloured, and the pupil blocked. There is a congenital coloboma of the iris downwards, and there are severe inflammatory changes of old standing. The eye was frozen and opened equatorially.

The pupil is much drawn towards the coloboma, and there is no opening in what is now the centre of the iris. The angle of the anterior chamber is completely closed by adhesion of the iris to the periphery of the cornea. The lens is *in situ*.

The retina is entirely detached except on one side where it forms a dense folded membrane reaching from the ora serrata to the optic disc. In it numerous cysts are developed. The folds of the retina enclose a small space which is the remains of the vitreous chamber. Both this and the sub-

retinal space are filled with coagulated albuminous material containing much blood.

There is an enormous coloboma of the choroid which occupies fully one half of the fundus. (Fig. 1.)

It involves the optic nerve, the head of which is deeply cupped. The whole of the sclerotic corresponding to the coloboma is thinned and bulged, owing doubtless to the tension under which the eye has for some considerable time been subjected.

The microscopical examination of the coloboma is interesting. The choroid can be traced running along the sclerotic in its usual position over a considerable area. It is thin and atrophic, and the pigment is irregularly scattered through its substance, showing that some inflammatory change has taken place.

As one approaches the coloboma the inflammatory changes become more marked and some hyaline bodies are seen (Fig. 2*b*), these are covered with hexagonal pigment; after this the pigment undergoes proliferation and is thicker and more distributed through the substance of the choroid. On coming nearer the coloboma the choroid is seen to cease, but a few processes extend forwards and are then lost in the tissue of the retina and sclerotic (Figs. 2*c* and 3*c*); a similar arrangement is seen on the opposite side of the coloboma.

The retina is greatly altered both in its disposition and also in its minute structure by inflammatory changes, so that some careful study is needed in order to find out its exact relations.

It appears, however, that it is entirely occupied in enclosing a space which is the remains of the vitreous chamber (Fig. 1*c*) but unlike most extensive detachments this one can hardly be termed complete, owing to the fact that the portion of the coloboma which is included within the remains of the vitreous chamber is actually lined with retina, which is in an extremely degenerate state and firmly adherent to the sclerotic (Fig. 3*a*).

The detached portion of the retina is very considerably

FIG. 1.

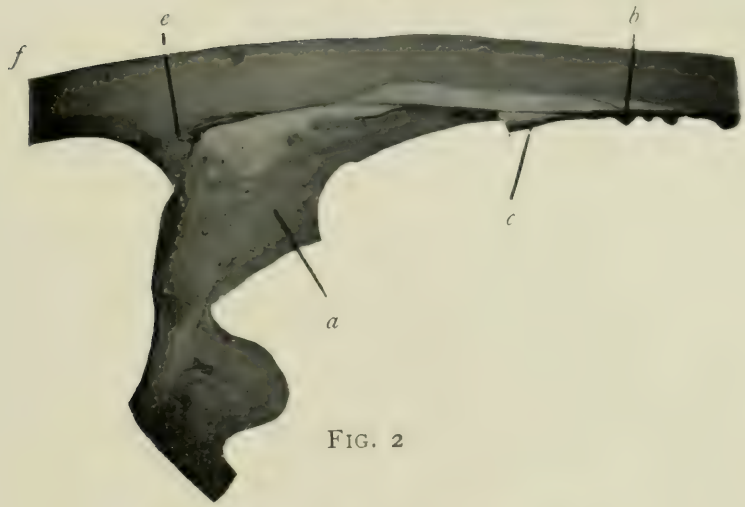

FIG. 2

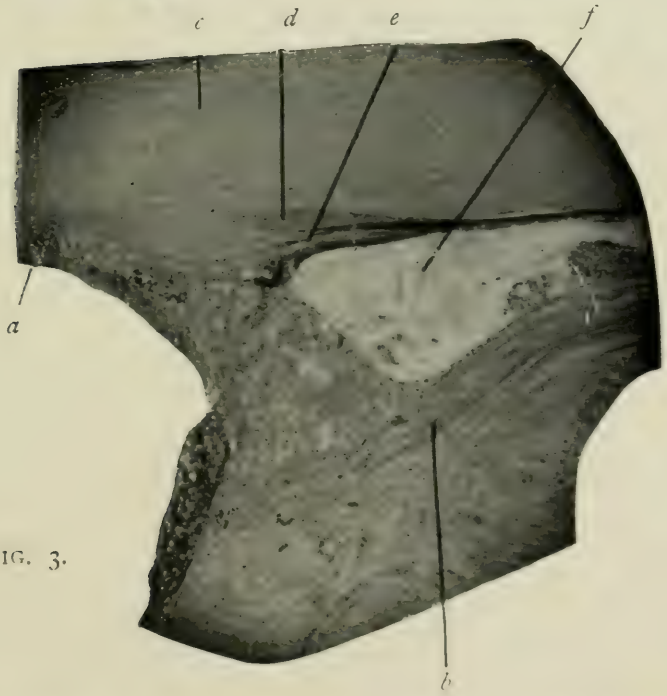

FIG. 3.

From photographs by the Author. *Engraved by Bale & Danielsson, Ltd.*

atrophied and far more fibrous than normal (Fig. 3*b*), while many of the minute elements which characterise it are either altogether absent, or else are so altered as to be unrecognizable.

The hexagonal pigment layer is almost entirely attached to the choroid, but in many parts of the retina isolated cells or groups of cells are adherent to it.

That portion of the retina which is attached to the sclerotic is chiefly characterised by its extremely honeycombed appearance, it is riddled throughout with small cysts which are filled with coagulated fibrinous material which stains slightly with eosin (Fig. 3*a*). The rest of the retina is in a somewhat similar condition, but the cystic spaces are nothing like so numerous.

On that portion of the retina forming one of the side walls of the remains of the vitreous chamber, the internal limiting membrane is completely stripped off, thus forming the inner boundary of a cystic space, the outer wall of which is formed by the rest of the retina.

In the portion of the vitreous chamber near the sclerotic, there is a good deal of organised exudation as well as numerous small round cells, while further away from the sclera it is filled with material similar to that which fills the smaller cyst-like spaces mentioned above.

As we trace the retina round we soon meet with another large cyst, which is apparently developed in the substance of the retina, but much nearer the outer than the inner side. Owing to the very degenerate state of the retina it is difficult to be sure of its exact position, but it is probably between the outer nuclear and outer molecular layers. This space is partially filled with exudation.

The anterior part of the eye shows still more remarkable changes. These are illustrated in part in Fig. 4.

In order to show this, the following dissection was performed:—

The eye, as described above, had been divided by an equatorial section. The cornea and sclerotic were removed from

the anterior half, leaving the iris, ciliary l
behind.

In order to see the suspensory ligament
had a coloboma was removed, and thus we
the ciliary processes from the front.

The position occupied by the coloboma
(Fig. 4*b*), for at that point the circle usually
ciliary body is incomplete, and instead of
assumes a pyriform shape.

From this point a median raphé runs do
apex of the coloboma where the choroid on e
meet (Fig. 4*c*), thus forming a uniting ban
iritic and choroidal colobomata. On cutting s
this raphé microscopical examination shows
ciliary processes which, instead of uniting i
way, have met together and become fused.
end of the bridge they cease, while at their
are continuous with the ciliary processes whi
proper place.

In order to produce this state of things
larger ciliary body than normal, for the eye is
as regards size, and is, in fact, myopic and e
no means be considered smaller than normal
the ciliary body only were developed, accoun
ent over-growth of the ciliary processes.

The tissue which fills up the space betwee
ally placed ciliary processes is unstriped m
seen under normal conditions, except that its

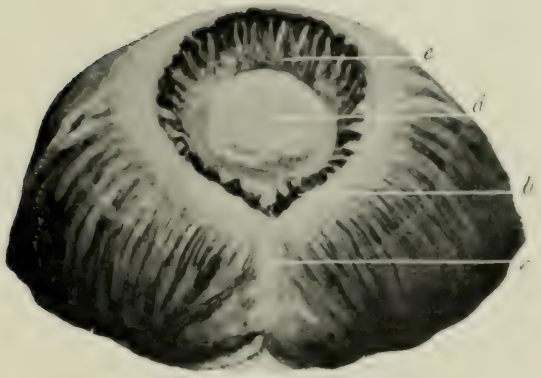

FIG. 4.

had a notch in it such as is frequently seen in these cases ophthalmoscopically, and it would have been extremely interesting to have examined the ligament and lens in detail, but this was impossible in the specimen.

As far as one could tell, the eyes had similar congential peculiarities, although the secondary inflammatory changes were far more marked in the left eye than in the right. In addition the retina of this eye is detached while the other is *in situ*.

The explanation of this condition is similar to that given for the ordinary colobomata of the iris and choroid, but in this case the developmental processes underwent some modification which lead to the defect in the choroid and iris, and possibly of the suspensory ligament and lens, as well as to an over-development of the ciliary body leading to the formation of the raphé where the processes from each side met.

A CASE OF TRAUMATIC PULSATING EXOPHTHALMOS; LIGATION OF THE RIGHT COMMON CAROTID ARTERY; PARTIAL CURE; REMARKS.

By Arnold Lawson, F.R.C.S.Eng.

I am indebted to Mr. John Tweedy and Mr. Henry Morris for their kind permission to publish my notes of this case.

Mrs. H——, æt. 41, a stout, healthy-looking woman, came to Mr. J. Tweedy's clinic, at the Royal London Ophthalmic Hospital, on March 16, 1897, complaining of severe headache and noises in the head.

Her history is as follows:—Eighteen months previously she had a blow on the back of her head, on the right side, the result of a fall downstairs. The blow was of sufficient force to render her unconscious for an hour. The accident occurred in the evening, and the following morning on waking up she found that she saw double. She went to Guy's Hospital, where she was admitted to Mr. Brailey's clinic. She then complained of occipital and frontal headache and double vision. The right eye was strongly converged, and could not be abducted beyond the midline, indicating paralysis of the 6th nerve.

R. V. = < 6/60. No improvement with glasses.
L. V. = 6/24 ℂ - 4·5 D. Sph. = 6/9.

She was ordered a plane ground glass for the right eye, and sph. – 3·5 D. for the left, and a mixture containing Pot. iod. and liq. hydrarg. perchlor. ordered three times a day.

She improved under this treatment, lost her headaches, and at her last visit to Guy's Hospital, nearly five months after the accident, it was noted that binocular vision had returned. This was on December 31, 1895.

A few weeks later she began for the first time to experience noises in her head, at first of a trifling character but constant, and increased on exertion. These noises grew gradually louder and louder, and towards the end of 1896 she noticed that the right side of her face was flushed, and that the right eye seemed more prominent than the left. The headaches began to return

about this time, and for the last three months before her first visit to Moorfields both noises and headaches had become very rapidly aggravated.

On examination the right side of the face was seen to be deeply flushed, especially at the upper part. The ocular conjunctiva was somewhat suffused, and was coursed by several tortuous dilated veins. There was marked proptosis of the right globe, but no paralysis of any of the ocular muscles. With the ophthalmoscope the retinal veins were seen to be enormously swollen and tortuous. No marked change was noted in the arteries; but the disc was blurred, and there were patches of effusion about the veins.

R. V. = < 6/60, $\bar{c}$ – 3 D. = 6/24. She had three diopters of myopic astigmatism, but no cylindrical correction improved her vision further. On placing the fingers in the hollow between the upper lid and orbital arch, the usual space was felt to be partially filled by a soft ill-defined pulsating mass, easily compressible, and imparting a marked rough thrill to the fingers. The pulsation and thrill were most marked in the neighbourhood of the supra-orbital notch, from which an enormously swollen pulsating supra-orbital vein could be easily traced over the forehead. The soft mass behind the lid was also evidently due to swollen pulsating veins. Another very large pulsating vein could also be seen running transversely outwards from the external canthus to join the anterior temporal vein. With the stethoscope a loud, rough, whizzing bruit was heard over the pulsating areas, but not elsewhere over the head. No pulsation was felt in the globe, and no pulsation was seen in the retinal veins. Pressure upon the carotid of the right side at once completely controlled pulsation and thrill.

The patient was ordered to rest and Pot. iod. grs. iij was ordered three times daily. She followed out the treatment for about six weeks, but grew much worse, and when seen at the end of that time she was evidently in great pain, and described the noises in her head as resembling the beating of a sledge-hammer. All the above-mentioned symptoms were pronouncedly worse, and it was decided to send her at once to the Middlesex Hospital, where she was admitted under the care of Mr. Henry Morris on April 30, 1897.

She was kept in bed for a fortnight to try what absolute rest in the horizontal position might effect, with a small ice-bag applied to the temple, a light diet, and Pot. iod. in small doses. She, however, got steadily worse, and her general health began to fail from the constant pain and want of sleep. About this time I again examined the fundus, and noted that there was a considerable amount of optic neuritis, and that the tortuosity had now extended to the arteries, especially to the smaller ones. A minute venous hæmorrhage was also apparent.

At the end of the fortnight further delay was obviously useless, so the patient was anæsthetised, and the right common carotid ligatured, opposite the cricoid cartilage.

Immediate and profound relief was experienced. On the morning after the operation all pain and noises in the head had left her, and all pulsation, bruit, and thrill had disappeared. From this time to the date of her leaving the Middlesex Hospital, June 17, the patient made an excellent and uninterrupted recovery. Three days after the operation I again examined the fundus, and found that a small hæmorrhage had occurred into the vitreous, but the veins were distinctly less congested, and the tortuosity of the arteries scarcely visible. The patient was kept in the horizontal position, with the head between sand-bags for rather more than three weeks after the operation. She was then allowed to sit up, and was finally allowed to move gently about the ward four weeks after the operation.

The following was her condition on leaving the hospital five weeks after the operation.

All subjective symptoms disappeared the day after the operation, and have not returned. The flushing of the right side of the face is scarcely noticeable; the proptosis and fullness under the orbital arch seem to have entirely disappeared, though there is a slight doughy resistance still to be felt. The dilated transverse vein, which was so prominent before the operation, has shrunk to a third of its former size, and the same applies to the supra-orbital vein. Pulsation and thrill have completely disappeared. The fundus is still rather obscured by vitreous opacities, but all neuritis has disappeared, and the edges of the disc look somewhat ragged and uneven. The tortuosity of the veins remains very marked, but they are much smaller and less congested. The arteries

appear quite normal. The only complication that ensued during convalescence was a slight attack of epistaxis, which occurred 10 days after the operation, and did not require any special treatment.

It will thus be seen that when the patient left the hospital the cure seemed complete.

I next saw the patient on July 7, three weeks after her discharge from the Middlesex Hospital. She then presented an almost complete relapse. All the old symptoms had re-asserted themselves with renewed vigour, with the exception of the noises in the head, which were only present in a modified degree. Since her discharge she had had two very severe attacks of epistaxis, and after each attack she felt considerably relieved for a while. She stated that as soon as she left the hospital and began to undertake home duties once more the old symptoms began at once to show themselves again. She was ordered to rest as much as possible, and to keep a piece of lint wet with lead lotion constantly applied.

I saw her again a week later. She had had another severe attack of epistaxis, two days after her last visit. This attack lasted about three hours, and since then she had felt much better. Her condition had once more considerably improved; no thrill could be felt, and the pulsation was not so heaving as before. The improvement, however, quickly disappeared, and on September 7 she was almost if not quite as bad as before ligature of the carotid. She stated, however, that she was feeling much better than during the previous week, when she had yet another attack of epistaxis more severe than any preceding it, and which had lasted almost continuously for two days. Nevertheless her condition seemed so grave about this time that the propriety of further operative measures was seriously considered; but Mr. Morris decided to wait a little longer.

However, since the date of the last attack of epistaxis, August 31, 1897, her condition has been one of steady improvement without any more relapses, and no further attack of epistaxis.

The matter may be briefly summed up by stating her present condition in October, 1898, when I last saw her.

There is complete absence of all subjective symptoms, headaches, noises in the head, &c. There is a small ill-defined swelling under the inner portion of the orbital arch, which yields a

faint expansile pulsation. No thrill can be felt, but there is a distinct bruit, though slight in nature. The right eye is slightly more prominent than its fellows, and the conjunctiva is rather congested. Pulsation can still be felt in the temporal vein above-mentioned and the supra-orbital vein, and both veins are a good deal enlarged. The right side of the face is still rather flushed. Ophthalmoscopic examination shows a very tortuous condition of the retinal veins, which, however, are nearly normal in size.

R. V. $\bar{c}$ −4 D. sph. = 6/24. Best improvement.
L. V. $\bar{c}$ −4·5 D. sph. = 6/9. „ „

Her general health is satisfactory, and she suffers no inconvenience, and were it not that there is practical evidence of the existence of the old trouble, which seems to present the possibility of a relapse at some future date, one would feel quite satisfied as to the result obtained.

The diagnosis of traumatic pulsating exophthalmos has now been placed upon a fairly certain basis. So that with our present knowledge, there can be no doubt that the case now recorded is one of arterio-venous communication at or near the cavernous sinus. Altogether the result of eight autopsies have been published. Six are collected by Sattler (Graefe-Seemisch, Band vi), one is recorded by Prichard (Bristol Med. Chir. Journal, vol. viii, 115), and one by Stuelp (Arch. of Ophth., vol. xxvi, 4). In six of these autopsies a rupture between the internal carotid and the cavernous sinus was definitely proved; in one case the presence or not of a rupture remained uncertain, whilst in one case only thrombosis of the cavernous and surrounding sinuses and ophthalmic vein was found. The last-mentioned case raises the question as to how far the train of symptoms may be due to simple thrombosis of venous sinuses. There are only three symptoms characteristic of arterial rupture, viz., (1) the sledge-hammer noises in the head; (2) the peculiar whizzing bruit; and (3) pulsation which is synchronous with the carotid pulse. All other symptoms, though compatible with arterio-venous communication, might be produced by

venous thrombosis alone. Even pulsation both ocular and extra-ocular might be evident, but such pulsation would *not* be synchronous with the carotid pulse.

The prognosis of these traumatic cases if left to themselves is still very uncertain; because the symptoms produced are of such a severe character that the patient nearly always applies for relief and is subjected to treatment. It is not fair to regard the matter from the standpoint of cases of idiopathic origin among whom a few cases of spontaneous cure have been reported, whilst others have continued in *statu quo* for years without treatment. The element of trauma of uncertain quality and quantity must greatly complicate the prognosis, and the call for active treatment often becomes much more urgent than in idiopathic cases. Speaking generally, the seriousness of the prognosis might perhaps be measured by the nature of the injury and the time that has elapsed between the injury and the origin of the symptoms of arterio-venous communication. When this period is very short, a few hours or a day or two, the injury has probably been very severe and possibly caused serious damage elsewhere; whereas if the symptoms, as in the present case, have not supervened until some months after the accident, we have only to fear the results of the arterio-venous communication, and the element of trauma may be practically discarded.

As regards symptoms this case presents two of some note.

1. An interesting feature is the attacks of profuse epistaxis. Similar attacks have occurred in other cases; but not nearly so commonly as one would expect, considering the free communication that exists between the nasal veins and the ophthalmic vein. In this case each attack has been attended with profound relief, and indeed her permanent steady improvement during the last 13 months has dated from the occurrence of her last and most severe attack. This may be accounted for by the temporary lowering of the blood pressure from the loss of blood, and the increase in its

coagulability from the same cause; both of which would favour the formation of a resistent clot. At the same time such attacks are not unattended by danger, as at least one fatal case of epistaxis in pulsating exophthalmos has been recorded.

2. The irregularity in the number of nerves implicated in these cases is interesting. In the present case only one nerve, the sixth, was involved; but in many, other nerves have also suffered, and in a few, total ophthalmoplegia interna and externa has been recorded. As would be expected from its position, the sixth nerve is the one most usually subjected to pressure. The escape of the third, fourth, and first division of the fifth nerve is probably due to their position on the outer side of the sinus where the bony wall is deficient, and they might be pushed aside. In a few cases the facial nerve has been paralysed, but this must have been the result of involvement of the nerve in a fracture of the base of the skull and could have no connection in any way with the arterio-venous communication in the cavernous sinus.

Lastly, a few words as to the radical treatment of traumatic pulsating exophthalmos.

Let us look at the statistics of ligature of the common carotid. Stuelp (Arch. of Ophth., vol. xxvi, 4, 97) has brought the list of recorded cases of ligature of the common carotid for this disease to 54. I have managed to collect seven further cases, including the present one, bringing the total to 61. Unfortunately, as Stuelp points out, the number is still too small to make accurate deductions, but there are one or two interesting points. On examination of the reports of these cases we are at first struck with the immense value of the treatment and the number of *cures* which it has effected, amounting to 33 or 54 per cent. On closer examination, however, the statistics will in measure lose their value; for, unfortunately, many cases reported as cures cannot strictly be regarded as such on account of the short time that has elapsed between the report and the cessation of treatment. Thus, as one example among many, in one case reported

in Sattler's list (Graefe-Sœmisch, Band vi) as a cure, only three weeks had passed since the ligature of the artery, and the last time that the patient was seen. Further, a certain number of cases are reported as cures which would be better classified under the heading of "partial cures," for some symptoms such as pulsation and bruit have persisted, and the patient, in that he was able to resume his former occupation, reported as cured. I venture to think that no case can be considered as cured until at least one year has elapsed since the time of operation; such time, in fact, as would give one reasonably sure grounds for considering the occlusion between artery and vein to be permanent; and further, that as long as any symptom, due to the persistence of such communication, such as bruit, &c., is evident even in a diminished degree, the disease can only be considered to be held in check and not cured. Examined by this light the records of ligature of the common carotid are not nearly so satisfactory as they at first appear. In Sattler's collection, 38 cases of ligature for traumatic rupture are reported, of which 16 are noted as complete successes. Of these 16 cases the report ends in three weeks, four weeks, six weeks, three months, four months, five months, and nine months respectively, whilst in three cases the date at which the patient is last seen is not mentioned. So that out of the so-called 16 complete successes the result may be said to be in complete doubt in 10, or nearly two-thirds of the total 16. Of the remaining 22 cases complete failure resulted in five cases; death, as a result of the operation, occurred in two cases, whilst improvement to varying extent was noted in 15 cases.

Ligature of the common carotid for this disease has been practically the one and only operative measure employed since Travers tied the artery for a case of idiopathic origin in 1805. In only two cases can I find a record of any other operative procedure and both were cases of idiopathic origin. In one case, Bronner (Lancet, 95, i, 1112) tied the internal carotid with successful result; whilst in the second case

Noyes (Trans. Am. Ophth. Soc., N. Y., vol. iii) performed a successful operation, which consisted in the ligature of all the large vessels in the neighbourhood of the swelling. One can only imagine that surgeons have been and still are perfectly satisfied that there is no better method than ligature of the common carotid; but in view of the large number of only partial successes, it might be well worth while to try a modification of this treatment in the shape of ligature of the internal carotid, which, I believe, might be attended with better results than ligature of the common carotid for the following reasons:—

1. The collateral circulation after ligature of the common carotid is maintained by three channels. Firstly and chiefly by the vertebrals and basilar through the circle of Willis: secondly, by the anterior communicating artery through the circle of Willis, and thirdly by a reflux of blood through the external carotid which has its circulation quickly re-established by free anastomoses across the neck. Very little stress is laid upon this latter channel in text-books, and indeed in some it is not even mentioned; but in these cases of pulsating exophthalmos, I believe it to be of great importance, because it is the only means by which the blood stream can be re-established in the normal forward direction between the origin of the vessel and the site of injury, with the exception of one or two unimportant communications about the tympanum and carotid canal. This reflux of blood through the external carotid must also be considerable, and probably established as quickly as any other channel of collateral circulation. If the internal carotid were ligatured, this means of collateral circulation will be cut off and thereby most, if not all of the direct arterial flow through the cavernous sinus. Moreover, any stream through the artery at this point would be produced by a reflux from its cerebral branches, and would be moving in a direction corresponding to the venous current; a condition which would be more favourable to the formation of clot between the injured artery and the venous channel than

would be the case if the arterial stream flowed in its usual direction.

2. If the common carotid is ligatured, a very large amount of blood is suddenly thrown back upon the other main channels of the head and neck. If the internal carotid only is blocked, the amount of blood thus thrown back will not be nearly so great, and the necessary dilation of collateral channels to restore the circulation will probably take place more gradually, which will favour the formation of a firmer clot at the site of injury.

3. On the ground that it is inexpedient to perform a major operation when a minor one will suffice, ligature of the internal carotid should be preferred to that of the parent trunk.

4. The technique of ligature of the internal carotid does not offer any special difficulty, as at its origin it is comparatively superficial, nor is there any greater danger attending its occlusion than a ligature applied to the common carotid.

Ligature of the internal carotid might also be supplemented by ligaturing the enlarged pulsating veins around the orbit, which are so commonly seen in these cases.

Finally, this case points out the necessity for prolonged rest in the horizontal position after ligation has been performed. In the case now recorded this treatment was most strictly carried out for nearly four weeks, and yet the subsequent history of the case seems to show that another fortnight in bed would have been well spent.

Ligature of the Common Carotid Artery for Traumatic Pulsating Exophthalmos. Cases not hitherto included in Statistics. Total collected cases reported by Stuelp (Arch. Ophth., vol. xxvi, p. 4) = 54 cases.

No.	Author.	Year.	Journal.	Sex.	Age.	Trauma.	Operation.	Result.
1	Walker	1888	Lancet, vol. xciv, p. 1	M.	37	Fall off horse	Lig. of right carotid	Cured with preservation of sight.
2	Prichard	1890	Bristol Med. Chir. Journal, vol. viii, p. 115	M.	30	Fall on back of head	Lig. of left carotid ..	Improved. Subsequent death one year later from rupture into ventricles. P.M. showed arterio-venous aneurism in cavernous sinus.
3	Puzey	1893	Lancet, vol. xciv, p. 1	F.	42	Fall into cellar	Lig. of right carotid	Cured with loss of sight.
4	Walker..	1894	Lancet, vol. xciv, p. 1	F.	33	Blow on left ear	Lig. of left carotid ..	Cured with preservation of sight.
5	Kibbe	1897	Arch. of Ophth., vol. xxvi, p. 1	M.	—	Head crushed between two heavy logs	Lig. of right carotid	Much improved.
6	Schimanowsky..	1897	Wjert. Ophth., vol. xix, p. 1	F.	52	Fall on back of head	Lig. of left carotid ..	Improved.
7	Lawson	1898	—	F.	41	Fall on back of head	Lig. of right carotid	Much improved.

SARCOMA OF THE UVEAL TRACT.

By C. Devereux Marshall, Curator.

In a paper published in the Royal London Ophthalmic Hospital Reports, vol. xiii, Pt. ii, in December, 1891, by Messrs. Lawford and Collins, all the cases of sarcoma of the uveal tract which could be obtained from the records of the hospital were tabulated and analysed. Records of 103 cases were found, and carefully worked out so as "to obtain evidence regarding the important question of prognosis in this affection."

This paper has proved to be of the greatest service to workers at this subject; but if such evidence is to be kept up to date, it is obviously the duty of those who have the opportunity of doing so, to continue to publish reports of the more recent cases, in addition also to still further following up those already published.

In order to fulfil these conditions, the following plan has been adopted :—

(1) All the patients who have been treated or examined at the hospital suffering from this disease since 1891, have been written to, unless evidence has already been obtained of their death. Of these there is a total number of 58.

(2) All the patients who were reported as being alive in 1891, and who were included in the previous paper have been written to, and in a large number satisfactory replies have been obtained either from them or their friends, and in many cases which have during the interval terminated fatally, the cause of death has been ascertained.

(3) An attempt has been made to search the death registers at Somerset House, for the purpose of ascertaining whether the names of those persons from whom no reply has been obtained appear therein, but owing to the extreme difficulty in locating the districts in which such people lived at the time of their death, and owing also to

the restrictions put upon those carrying out what is termed "a general search" by the authorities, together with the probability of obtaining unsatisfactory results, especially with regard to the cause of death in those traced, it has been decided, on the advice of a competent authority, to abandon the search as being, under existing circumstances, impracticable.

(4) The further information concerning those recorded in the previous paper will first be stated, followed by an analysis of the 58 cases, which form the basis of this present report.

(5) All particulars relating to cases in the previous paper which are not to be found here, will be seen on reference to the original. Those which have occurred since 1891 will be dealt with in detail.

After-History of the Cases reported in the Previous Paper.

Of the 103 cases in the 1st series, 40 were dead in 1891, and since then we have found that nine others are dead, and two besides these had, when last heard of, recurrence, so that we have now 49 cases in which we know that death has occurred, and if we include the two other cases, this will make 51, besides 46 others which are unable to be traced.

In 1891 the following after-history of cases was obtained:—40 or 50·63 per cent. were dead; 39 or 49·36 per cent. were alive, and in the remaining 24 cases the after-history was not obtainable.

Since that date we find the following:—

Of the 39 patients who were known to be living, six are still alive, Nos. 35, 37, 64, 85, 89, 95.

Ten are dead, Nos. 58, 75, 84, 87, 88, 93, 97, 98, 99, 100, while there are two cases which were noted shortly after the paper was published as suffering from recurrence; these are Nos. 67 and 81. In the former there was local and general recurrence, and it is certain the patient did not live many weeks, while in the latter the patient had, when last heard

of, an enlarged liver, with nodules in the skin, so that we may fairly assume that both these died from spread of the disease, for which the eye was removed.

Patients still Alive.

Of the 103 cases, there are known to be but six patients living at the present time: they are the following:—

No. of case.	No. of years alive after operation.
35	17
37	16
64	$11\frac{10}{12}$
85	$9\frac{9}{12}$
89	$9\frac{6}{12}$
95	$8\frac{8}{12}$

6 cases.

The number of cases which cannot be traced is now 45 instead of 24, which was the number unaccounted for in 1891.

If we now examine the alleged cause of death in the ten cases in which a fatal result is recorded, together with the two in which we assume that death has taken place, we find as follows:—

No. of case.	No. of years alive after operation.	Alleged cause of death.
58	Unknown	Cause of death unknown.
75	Unknown	"Cancer of stomach."
84	About 4 years	General recurrence in liver, skin, &c.
87	$3\frac{7}{12}$	Recurrence in abdomen.
88	$2\frac{7}{12}$	"Died of cancer."
93	8	Cause of death unknown.
97	$6\frac{5}{12}$	Died of bronchitis. No recurrence.
98	3	"Died of cancer."
99	$4\frac{10}{12}$	Died of general recurrence.

No. of case.	No. of years alive after operation.	Alleged cause of death.
100	7	Died of cystitis, and hæmaturia, "probably due to malignant growth." No autopsy made.

Of the two we assume to be dead, the probable length of time that the patient lived, and the probable cause of death, was as follows:—

No of case.	No of years alive after operation,	Probable cause of death.
67	$11\frac{5}{12}$	Local and general recurrence.
81	About a year	General recurrence, skin, liver, &c.

If we add these 12 fatal cases to the list of 40 previously published, we find that 52 cases died out of 58 cases traced. Of these, 34 died of recurrent disease, giving a percentage of 58·62, this being a good deal larger than was found seven years ago, when it was 32·9 per cent.

These figures will again be referred to later on, when the more recent cases are taken.

Cases not previously Published.

We will now consider the cases which have occurred at this hospital since 1891, and we shall thus have 161 cases, extending over a period of 27 years.

Sex of Patients.

Of the 58 cases not previously reported, there were 24 males, 31 females, and three in which the sex was unknown. This gives a percentage of 41·38 males and 53·45 females, and 5·17 unknown.

The higher proportion of females is unusual. In the previous paper 57·28 per cent. were males, and 42·71 per cent.

were females, and in addition the authors go on to state of 259 cases recorded by Fuchs, there was a slight preponderance of males, while in only one paper by Martin containing a record of 43 cases, were there more women than men, 29 having occurred in women and 14 in men.

Age of Patients.

The following table shows in decades the ages of the patients :—

Years.	Males.	Females.	Total.
Between 20 and 30 there were	1	0	1
„ 30 „ 40 „	2	5	7
„ 40 „ 50 „	3	3	6
„ 50 „ 60 „	7	9	16
„ 60 „ 70 „	5	12	17
„ 70 „ 80 „	3	2	5
Age unknown	...	...	6
	21	31	58

The average age of the 52 known cases is 54·63. The youngest patient in this list was a man aged 28 years, No. 48, and the oldest a woman aged 74, No. 26.

Eye Affected.

Right eye	27
Left eye	28
Unknown	3
	58

Tension at the time of the operation, and the condition of the angle of the ant. chamber :—

Tension.	No. of cases.	Condition of angle.
T − 3	4	Closed in all.
T + 2	12	Closed in 11, narrowed in 1.
T + 1	6	Closed in 5, narrowed in 1.
T +	4	Closed in 1, narrowed in 2, not stated in 1.
T ? +	1	Open.
T n.	20	Open in 16. Closed in 1. Doubtful 1. Not stated 2.
T ? −	1	Not stated.
T −	2	Angle open in both.
T − 1	3	Angle open in all.
Not stated	2	
Globe solid with growth	2	
Globe bony and full of growth	1	
Total ...	58	

The following table is an analysis of the cases classed according to the tension of the eye at the time of operation, and the situation of the growth :—

Tension of eye.	Cases in which the ciliary body is involved.	Cases in which the choroid is affected without the ciliary body.	Total.
T + 3	2	2	4
T + 2	..	12	12
T + 1	3	3	6
T +	..	4	4
T + ?	..	1	1
T n.	10	10	20
T ? −	1	..	1
T −	1	1	2
T − 1	3	..	3
	20	33	
2 cases in which the globe was solid with growth, in both of which the ciliary body was affected 1 case in which the eye had bone in it 2 cases in which the tension was not stated			5
			58

We see from the above table that of the cases in which the tension was above normal, there were 22 in which the choroid only was affected, and only 5 in which the ciliary body was involved in the growth.

There was normal tension in an equal number of cases, while the tension was — in five cases where the growth involved the ciliary body, and in only one case in which the choroid only was affected.

The following is a summary of the table on tension :—

	Tumours of choroid.		Tumours of ciliary body.	
	Actual number.	Percentage.	Actual number.	Percentage.
Increased tension	22	66·66	5	25·00
Normal tension	10	30·30	10	50·00
Diminished tension ..	1	3·03	5	25·00

This again is confirmatory of a point brought out by the author in a paper before the Ophthalmological Society and published in the Transactions, vol. xvi, 1896, in which 100 cases of intraocular tumours were taken of all sorts, and it was found that, although the figures differed somewhat from the above, yet the general conclusions hold good, viz., that in growths which do not affect the ciliary body there is far more likelihood of the tension being increased than in cases in which the ciliary body is involved; in fact the tension is on a distinctly higher level in the former than it is in the latter.

The operations performed in these cases were as follows :—

In 56 cases ..... Enucleation of the eye only.
In 2 „ (50 and 57) Enucleation with subsequent extirpation of the contents of the orbit within a few days.

The following cases are known to have been alive for the number of years stated :—

No.					Years.
3 has lived since the excision				.....	7
4	,,	,,	,,	.....	7
5	,,	,,	,,	.....	7
7	,,	,,	,,	.....	$5\frac{4}{12}$
8	,,	,,	,,	.....	$6\frac{3}{12}$
9	,,	,,	,,	.....	$6\frac{1}{12}$
13	,,	,,	,,	.....	$5\frac{5}{12}$
17	,,	,,	,,	.....	$4\frac{1}{12}$
18	,,	,,	,,	.....	3
19	,,	,,	,,	.....	3
25	,,	,,	,,	.....	$3\frac{1}{2}$
27	,,	,,	,,	.....	$2\frac{8}{12}$
29	,,	,,	,,	.....	$2\frac{8}{12}$
31	,,	,,	,,	.....	$2\frac{9}{12}$
34	,,	,,	,,	.....	$2\frac{5}{12}$
35	,,	,,	,,	.....	$2\frac{5}{12}$
36	,,	,,	,,	.....	$1\frac{2}{12}$
37	,,	,,	,,	.....	2
38	,,	,,	,,	.....	$2\frac{1}{12}$
41	,,	,,	,,	.....	$1\frac{9}{12}$
42	,,	,,	,,	.....	$1\frac{10}{12}$
44	,,	,,	,,	.....	$1\frac{1}{2}$
46	,,	,,	,,	.....	$1\frac{2}{12}$
47	,,	,,	,,	.....	$1\frac{1}{12}$

There are 11 cases which have been operated upon within the last year, and these, as far as is known, are still alive: that is to say, there are 35 cases known to be alive out of the 58 in this series.

Of the 35 cases which are alive there are 11 which have remained free from recurrence for three years and upwards; the longest interval being in Cases 3, 4, and 5, in which a period of seven years has elapsed since the eye containing the tumour was removed.

We should, however, be careful to avoid drawing an arbitrary line such as a three years limit of recurrence, for cases do recur at a greater interval of time than this, such as is seen in Case 67 (first series), where the patient died of recurrence four years after the removal of the eye.

Causes of Death.

Six cases out of the total of 58 in this series are known to have died of recurrence; these are:—

Nos. 22, 23, 28, 30, 32, 45.

There are, however, 12 cases the fate of which is unknown. If we exclude these the percentage of deaths from recurrence amounts to 13·04.

The percentage found by Lawford and Collins was 32·9 out of the 79 cases which were traced, but since then the percentage has risen to 58·62.

Of the six cases in which there is no evidence that the patients died of recurrence there are two cases in which the cause of death is not known. These are Nos. 1 and 20.

The cause of death in the remaining four is given as follows:—

No. 14 died of "heart disease." Date unknown.
,, 21 ,, "senile decay." 3 years after operation.
,, 26 ,, "inflammation of kidneys." ? Metastatic growth. $1\frac{9}{12}$ years after operation.
,, 39 ,, "strangulation of the bowel," nine months after operation.

The following 34 cases are known to be alive:—

Nos. 3, 4, 5, 7, 8, 9, 13, 17, 18, 19, 25, 27, 29, 31, 34, 35, 36, 37, 38, 41, 42*, 44, 46, 47, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58.

Of these Nos. 18 and 46 have Bright's disease, and they both have in addition albuminuric retinitis.

Situation of Recurrence.

Of the six cases in which there was known to be recurrence, No. 22 died in about $2\frac{1}{4}$ years of recurrence all over the body. No. 23 had local and general recurrence, and died five months after the eye was removed. No. 28 had recurrence "in the bone of her arm, which caused it to break,

* Patient has since died in S. George's Hospital of Sarcoma of the Liver, &c. The case is fully reported by Dr. H. D. Rolleston in The Lancet, May, 13, 1899.

necessitating amputation at the shoulder, after which the disease spread all over the body," and she died 14 months after the eye was removed. No. 30 died in S. Bartholomew's Hospital in February, 1897, 13 months after the removal of the eye, with recurrence in every part of the body, including a local recurrence in the orbit. She also had melanuria. No. 32 died in $1\frac{1}{3}$ years with recurrence in the lungs, abdomen, one of the bones of his leg (which nearly caused it to break), and all over the body. No. 45 died $1\frac{7}{12}$ years after the removal of the eye of "cancer of the liver." She also had a recurrence in the scalp, the nature of which was proved by microscopic observation after its removal during life. There was no local recurrence in the orbit.

Thus we see that two out of the six cases are known to have had local recurrence.

Six or exactly half (50 per cent.) of the cases which are known to be dead died of recurrence, and of these two had local and general recurrence, or 33·33 per cent., and in one of them (No. 23) the sclera had been perforated and the growth extended into the orbit before the eye was removed; while four had no local recurrence—that is to say, 66·33 per cent.

The proportion of local recurrence here is much larger than that found by Lawford and Collins, which was 8·86 per cent.

History of previous Injury.

In six cases a history of previous injury was obtained. Nos. 1, 22, 29, 50, 55, 58; while in No. 57 the affected eye is known to have been defective and divergent for many years through an unascertained cause.

Of the injured eyes No. 50 is the only one in which there is a definite history of a severe blow or wound. This was caused 30 years before removal by a blow from a skipping rope. The eye had remained quiet until a few months before removal, though quite blind. In addition there was a dense mass of bone in the choroid.

In No. 1 there is a history of a blow on the eye 30 years

ago with a gas pipe, followed by erysipelas, but from this the patient quite recovered, and the sight had failed for only three months.

No. 22 received a blow 12 years ago, but as the sight failed only one month before removal, it is not likely that any connection exists between the two events.

No. 29 had a slight injury four months before removal.

No. 55 was struck in the affected eye two years before the excision, immediately after which the sight failed. The patient, however, attended the hospital on account of pain which had been present for two weeks.

No. 58 had a slight injury to the eye 14 years before excision, but from this it quite recovered. The sight had, however, been failing for three years, and the eye had been quite blind for one year.

There is a history of injury in 10·34 per cent. of these cases, but in most of them the injury was probably a coincident and had nothing really to do with the malignant disease from which the patient suffered.

Considering, however, the fact that a lost eye containing bone is liable to become the seat of a malignant growth, and that frequently owing to the condition present the tumour is not able to be seen until it has existed for many months, one should not be too conservative in the treatment of blind eyes which after months or years of quiescence become painful. It will be found that in a large number of cases the secondary symptoms are due to malignant growths.

Family History.

With regard to the family history one cannot say very much, chiefly because of the somewhat unreliable statements which patients frequently make concerning their deceased relatives.

However, in a very large number of cases the history, so far as is obtainable, is absolutely negative. There are, however, four cases in which there is a history of malignant disease in the family, though in most of them there is no means of determining the nature of the "tumours" or

"cancers" from which the patients are supposed to have succumbed.

In No. 7 the father of the patient is said to have died of "cancer in the back."

In No. 18 the grandmother of the patient was "blind from tumour in the eye," and her (the patient's) mother had a "tumour of the neck."

In case 37 the patient's father died of "cancer of the bladder," and in No. 42 we are told that "there is a strong family history of cancer."

From such meagre information as this one cannot say that the tendency to sarcoma of the choroid is very marked in any of the families.

There is, however, one case which has been traced, and, as the family history is reliable, and as the disease affected many members of the family, it will be interesting to mention it here, especially as it completes a case which was included in the previous paper.

It is Case No. 99 in that series.

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of Patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
1. 3459	W. T.	27.6.91	Thomas Goulding	R.	56	Sight failing 3 months without pain.	Unknown
2. 3488	W. L.	28.8.91	(Middlesex Hospital case)	L.	..	..	..

On reference to this it will be seen that the patient is a daughter of Case No. 2, who had the left eye removed, together with some of the orbital contents for a melanotic sarcoma of the choroid which had perforated the sclerotic and was affecting the orbit. She became an in-patient at S. Bartholomew's Hospital suffering from abdominal recurrence, and she died about two years after the eye had been removed.

The best thanks of the author are due to Messrs. Nettleship, Tweedy, Lang, Morton, Rockliffe (Hull), Johnson Taylor (Norwich), Collier Green (Derby), and to the late Mr. Hodges, of Leicester, all of whom have provided cases which are included in this report, either from their private practice or from their practice at hospitals other than Moorfields to which they are attached, while most of those mentioned have spared no pains in finding out the subsequent history of their cases for the present paper. Mr. N. C. Ridley (Leicester) has also kindly given me some later notes of the cases which occurred in the practice of Mr. Hodges.

SARCOMA OF THE EYEBALL.

uration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
ıd, but date and use of death unnown.	Melanotic tumour involving base of iris, ciliary body, and choroid as far back as equator. Its cells are spindle-shaped, and the optic nerve is unaffected.	Tension normal.	..	Mother died of "internal cancer," aged 75 years. Affected eye injured with a gas pipe 30 years ago, followed by erysipelas. The sight quite recovered from this.
.. ..	Deeply pigmented growth reaching from base of iris to equator and inwards to middle of globe. It is a pigmented spindle cell sarcoma.			

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence
3. 3493	R. M. G.	2.9.91	Daniel Thompson	R.	55	Sight failing 1 year. Blind 4—6 weeks. Pain 1 week.	..
4. 3509	A. S. M.	22.9.91	Mr. P. (Private patient)	R.	..	.. ..	..
5. 3525	Mr. Johnson Taylor	15.10.91	Mrs. G. (Private patient)	R.	60	..	..
6. 3534	Mr. Collier Green	26.10.91	Ellen T—— Case occurred at Derby General Infirmary.	L.	55	Sight failing for several weeks. Blind and very painful 1 week.	..
7. 3697	A. S. M.	3.5.92	Catherine Chapman	R.	30	Sight failing 1 month. No pain.	..
8. 3710	J. T.	17.5.92	Emily Chapman	L.	58	Sight failing 9 years. Blind and painful 1 year.	..
9. 3766	E. N.	30.7.92	John Dean	L.	60	Sight failing 1 year.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
live and in good health 7 years after excision.	Pear-shaped melanotic sarcoma of choroid. O. N. is encroached upon, but not involved. Tumour is a mixed round and spindle cell growth not deeply pigmented.	T + 2, angle closed.	..	Eye injured with a piece of wood 3 weeks before excision. F. H. negative.
live and in good health 7 years after excision.	Mushroom-shaped growth with broad base nearly reaching O. N. behind and O. S. in front. Slightly pigmented small spindle cell sarcoma. Sclera somewhat involved.			
live and in good health 7 years after excision.	Two tumours in ciliary region on opposite sides, each size of a pea. Small spindle cell sarcomata containing pigment.	T. normal.	..	No known history of tumour in the family.
.. ..	Mushroom-shaped growth of choroid extending from close above O. D. to equator.	T. + 2, angle closed.		
live and well $5\frac{1}{12}$ years after excision.	Tumour reaches from ciliary body to within 4 mm. of O. D. O. N. healthy. Slightly pigmented spindle cell sarcoma.	T. normal, angle open.	..	Father died of "cancer in the back." No history of injury or previous eye disease.
live and well $6\frac{3}{12}$ years after excision.	Melanotic mixed cell sarcoma arising from choroid around O. D.	T. normal, angle open.	..	F. H. negative. No history of injury or previous eye disease.
live and well $6\frac{1}{12}$ years after excision.	Unpigmented sarcoma growing from choroid on inner side, reaches from O. D. nearly to O. S. Tumour involves ciliary artery and extends into it.	T. normal, angle open.	..	Slight injury 2 years ago.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
10 3812	A. S. M.	17.9.92	Sarah Jackson	L.	56	Sight failing 9 months. Blind 4 months. Painful 6 weeks.	..
11 3854	J. B. L.	16.10.92	George Jackson	R.	37	Eye blind 15 months. Painful 1 week.	..
12 3933	J. C.	28.2.93	Eliza Clements	L.	46	Sight failing without pain for 2 months.	..
13 3962	Mr. Johnson Taylor	9.3.93	Sarah R. (Private patient)	R.	53	..	..
14 4007	W. L.	16.5.93	Charles King	L.	62	Sight failing 1 year. Blind 5½ months. Pain 3 weeks.	..
15 4029	E. N.	17.6.93	Miss M. J. B.	R.	63	Flickering before eyes 1 month.	..
16 4141	J. C.	27.10.93	Sarah Haddon		52	Sight failing 6 months without pain.	..
17 4354	W. T	9.7.94	Helen Chalcroft	R.	63	Sight failing 1 year with occasional attacks of pain.	..
18 4428	A. S. M.	2.10.94	Annie Kiff	R.	30	Sight failing for 20 months. No pain.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence	Family history, remarks, &c.
Not able to be traced.	Slightly pigmented sarcoma growing from the outer half of the choroid.	T. + 2, angle closed.	..	F. H. negative.
..	Large pigmented growth on outer side of globe reaching nearly forwards to lens. Retina detached elsewhere.	T. +, angle narrowed.	..	F. H. negative. Not able to be traced.
..	Melanotic spindle cell sarcoma reaching from ciliary body to equator.	T. normal, angle open.	..	F. H. negative. No history of injury or of previous disease. Not able to be traced.
Alive and well $5\frac{5}{12}$ years after excision.	Melanotic sarcoma of posterior part of choroid close to O. D. and Y. S. Small spindle cell growth	T. normal, angle open.	..	F. H. unknown.
"Died some time ago of heart disease."	Small spindle cell melanotic sarcoma of choroid reaching from O. D. to equator. O. N. not affected.	T. + 2, angle narrowed	..	F. H. negative. No history of injury or of previous disease.
..	Melanotic sarcoma reaching from ciliary body to O. N. Small round and spindle cell growth.	T. ? −	..	F. H. unknown. Not able to be traced.
..	Slightly pigmented small round and spindle cell sarcoma growing from choroid.	T. +, angle narrowed	..	F. H. negative. No previous injury or disease. Not able to be traced.
Alive and well $4\frac{1}{12}$ years after excision.	Small round and spindle cell melanotic sarcoma of choroid. O. N. healthy.	T. normal, angle open.	..	F. H. negative. No previous injury or disease.
Alive 3 years after excision, but has albuminuric retinitis in the other eye.	Mixed round and spindle cell pigmented sarcoma of choroid reaching from near ciliary body to O. D.	T. full, angle greatly narrowed.	..	Grandmother blind from tumour of the eye. Mother has tumour of neck.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
19 1442	R. M. G.	17.10.94	Alfred Gibbons	L.	43	Sight failing 4 years. No pain.	..
20 1477	A. Q. S.	3.12.94	Mary Dorrington	R.	61	Sight failing 2 years. Pain 1 month.	..
21 1481	Mr. Hodges, Leicester Infirmary.	6.12.94	John Bates	R.	73	Eye blind for 16 years. Pain 4 months.	..
22 1483	W. T.	13.12.94	Edward Rittman	L.	45	Sight failing 1 month.	About $2\frac{1}{4}$ years.
23 1486	A. S. M.	14.12.94	Annie Carter	R.	40	Sight failing 3 years. Pain 3 weeks.	A few weeks.
24 1 O P.	A. S. M. G. N. Hosp.	12.2.95	..	..	..	..	..
25 1561	A. Q. S.	21.3.95	Harriet Godfrey	R.	61	No symptoms were noticed by patient.	..
26 1681	A. S. M.	30.8.95	Theriza Horn	L.	74	Eye affected "some time." Occasional pain.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive 3 years after excision, but in bad health. Suggestive of abdominal recurrence. No local recurrence.	Unpigmented sarcoma close to O. N. Sclera much thinned and almost perforated. It is a small round cell growth.	T. normal, angle open.	..	F. H. negative. No injury or previous disease.
1 year	Melanotic sarcoma, nearly filling globe.	T. +2 angle closed.	..	F. H. negative. Cause of death unknown.
3 years. Died of "senile decay." No recurrence.	Globe full of a dense round cell melanotic sarcoma, which has perforated sclerotic near equator. Large extra-ocular mass nearly as large as the eye.	Globe solid.	..	F. H. unknown.
About 2¼ years ..	Rather large mottled sarcoma of choroid. It is a round and spindle cell growth.	T. normal, angle open.	Liver, lungs, and probably other viscera.	F. H. negative. Blow 12 years ago; probably not a severe injury.
5 months	Large flattened melanotic sarcoma of choroid which has perforated sclera near O. N. Large mass in orbit.	T. +3, angle closed.	"Cancer developed all over her including the orbit."	Father died of cancer. No injury or previous eye disease.
..	Round and spindle cell melanotic sarcoma, extending from O. D. to about equator.	Angle open.		
Alive and well 3½ years after excision.	Sarcoma the size of a pea in ciliary region with a large cyst developed in it.	T. normal, angle open.	..	F. H. negative.
19 months	Melanotic sarcoma in ciliary region pushing the iris forwards.	T. normal, A. C. shallow.	..	F. H. negative. Patient died in May, 1897, from "inflammation of the kidneys."

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
27 590 P	Mr. Rockliffe (Private case).	22.10.95	Mr. S. P. H.	L.	42	Sight failing for a "few weeks."	..
28 4748	W. L.	18.11.95	Emma Topsfield	L.	64	Eye weak for 4 to 5 years. Occasional pain.	Unknown
29 4765	R. M. G.	7.12.95	Emily Gooding	L.	60	Sight failing 4 to 5 months.	..
30 4779	E. T. C.	26.12.95	Eliza Thacker	R.	70	Sight failing 2 months. Blind and painful 3 weeks.	6 weeks
31 4782	A. S. M.	31.12.95	Mary Nash	R.	33	Sight failing 6 months.	..
32 4795	J. B. L.	8.1.96	Charles Piper	R.	71	Sight failing for many months. Blind 1 year. Pain 6 weeks.	Unknown
33 750.P	J. T. Case at U. C. H.	28.2.96	John Reed	..	..	..	..
34 4814	W. T.	9.3.96	Mary Radley	L.	55	Sight failing 3—4 months.	..

Sarcoma of the Eyeball.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive and in good health $2\frac{8}{12}$ years after excision.	Unpigmented sarcoma of choroid composed of mixed round and spindle cells, and extending from O. N. to equator.	T. normal, angle open		
14 months	Melanotic sarcoma reaching from ciliary body to within 3 mm. of O. D. Mixed round and spindle cell growth.	T. −, angle open.	General recurrence all over body.	Before death the arm fractured and was amputated "for cancer."
Alive and well $2\frac{8}{12}$ years after excision.	Unpigmented small round cell sarcoma growing from ciliary body and reaching to equator.	T. normal, angle open.	..	Slight injury 4 months before excision.
13 months	Melanotic sarcoma extending from iris to within 15 mm. of O. N. O. N. healthy.	T + 3, angle closed.	Local and general recurrence in every part of body.	Patient had melanuria, and died in St. Bartholomew's Hospital. F. H. negative.
Alive and well $2\frac{9}{12}$ years after excision.	Melanotic sarcoma reaching from ciliary body to posterior pole of eye.	T. −1, angle open.	..	No injury. F. H. negative.
16 months	Melanotic sarcoma reaches root of iris and half fills globe; this has given way on one side, and there is a large extra-ocular mass.	T. + 3, angle closed.	General recurrence May, 1897.	Patient had recurrence in one of the bones of the leg which nearly caused it to break, as well as all over the body.
..	Unpigmented sarcoma occupying region of O. N.	Angle closed.		
Alive and well $2\frac{5}{12}$ years after excision.	Melanotic sarcoma in ciliary region about the size of a split pea. Small round cell growth.	T. + 1. Angle open on one side, closed on the other.	..	F. H. negative. No previous injury.

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
35 4851	J. T.	17.3.96	Jane Dalgleish	R.	37	Sight failing several years. Blind 2 months.	..
36 4928	E. N.	11.6.96	James Campbell	R.	55	Sight failing 1 year. Blind 3 weeks.	..
37 4991	A. S. M.	18.8.96	Robert Woodcraft	R.	61	Sight failing 5–6 weeks.	..
38 4969	R. M. G.	25.7.96	Edmund Howard	L.	63	Sight failing 3 years. Blind 5 months. Pain 1 month.	..
39 4993	E. T. C.	20.8.96	James Wastell	R.	69	Sight affected for 8 months.	..
40 137 O.P.	Mr. Rockliffe. Case from Hull.	31.10.96	Sarah B.	L.	50	Acute glaucoma for 2—4 weeks.	..
41 5048	E. T. C.	12.11.96	Mary Jenkins	R.	49	Sight failing for 8 months. No pain.	..
42 137 O. P.	A. S. M.	15.11.96	Mr. B.	L.	About 50	Sight failing for 7 weeks.	..
43 141 O. P.	Mr. Rockliffe Case from Hull.	21.11.96	.. ..	..	..	.. ..	..
44* 5113	W. L.	18.2.97	William Dumsday	R.	57	Sight failing 3 months.	..

* See footnote, page 59.

SARCOMA OF THE EYEBALL.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
Alive and well $2\frac{5}{12}$ years after excision.	Melanotic sarcoma reaching from near O. N. to equator.	T. normal, angle open.	..	F. H. negative. Patient not very strong.
Alive and well $1\frac{2}{12}$ years after excision.	Small round cell melanotic sarcoma growing from region of equator.	T. +.	..	F. H. negative. No history of injury.
Alive and well 2 years after excision.	Slightly pigmented small round cell sarcoma reaching from ciliary body to equator.	T. −, angle open.	..	Father died of cancer of the bladder. No history of injury or previous eye trouble.
In good health $2\frac{1}{12}$ years after excision.	Unpigmented spindle cell sarcoma which is invading the sclerotic at one place.	T. +2.	..	F. H. negative. No previous eye affection.
9 months	Large melanotic sarcoma reaching from ciliary body to O. D. Small round and spindle cell growth.	T. normal, angle open.	..	F. H. negative. No injury. Patient died of "strangulation of the bowel," May, 1897.
.. ..	Mushroom-shaped melanotic sarcoma growing from choroid.	T. +2, angle closed.	..	Unable to be traced.
Alive and well $1\frac{9}{12}$ years after excision.	Slightly pigmented sarcoma; it is thin and flat.	T. normal, angle open.	..	F. H. negative. No injury or previous eye disease.
Alive and well $1\frac{10}{12}$ years after excision.	Melanotic sarcoma extending from ciliary body to within 5 mm. of O. D.	T. −1, angle open.	..	Strong family history of cancer.
.. ..	Melanotic sarcoma of ciliary body and of choroid.	Angle closed.	..	Unable to be traced.
Alive and well $1\frac{1}{2}$ years after excision.	Melanotic sarcoma extending from edge of O. N. to equator.	T. normal, angle open.	..	F. H. negative. No injury or previous disease.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	
45 5127	W. L.	1.3.97	Caroline Slade	L.	67	Sig l M
46 5214	E. N.	19.6.97	Jane Brown	R.	65	Sig y c
47 5231	E. N.	10.7.97	James Cooper	L.	56	Sig n p
48 5232	E. T. C.	12.7.97	Frank Phillips	L.	28	Sig n f
49 5309	E. N.	16.10.97	Rosana Bentley	L.	60	De l P

SARCOMA OF THE EYEBALL.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	F
Died 1 $\frac{1}{2}$ years after excision.	Large round and spindle cell melanotic sarcoma filling two-thirds of globe. Ciliary body not affected.	T. +1, angle closed.	Liver and scalp.	F. H inju cur
Alive and well 1 $\frac{2}{12}$ years after excision.	Large melanotic sarcoma composed of small round cells extending from O. N. on one side to within 3 mm. of the ciliary body on the other.	T. +2, angle closed.	..	F. H inju albu
Al ve and well 1 $\frac{1}{12}$ years after excision.	Slightly pigmented small round cell sarcoma. Ciliary body not involved.	T. −, angle open.	..	F. H inju eye
..	Large melanotic sarcoma with round and spindle cells; it is growing from the choroid and just reaches the ciliary body.	T. +2, angle closed.	..	F. H tie tra
n good health 10 months after excision.	Two nodules of growth outside sclerotic near superior oblique insertion. Mottled growth on disc connected with s ı all growth outside; also flat mass extending from this, 0·5 mm. thick occupying posterior third of eyeball. Slightly pigmented round cell sar-	T. +2, angle closed.	..	F. H inj is fus to t ret opl

SARCOMA OF THE EYEBALL.

No. of case and No. in Hosp. Reg.	Surgeon	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
51 5380	R. M. G	19.1.98	Mary Jerrell	L.	67	Two or three attacks of acute glaucoma during 8 months.	..
52 5424	J. B. I.	2.4.98	Thomas McCombe	L.	73	Sight failing 7 years. Blind 5 years. Old iritis.	..
53 5481	A. Q. S.	28.6.98	Charles Pain	L.	34	Sight noticed to be failing 3 weeks.	..
54 5488	J. T.	5.7.98	Louisa Cheshire	L.	57	Sight failing 10 months. Blind 7 months.	..
55 5491	W. T.	7.7.98	Susan Sheppard	R.	39	Sight failed 2 years ago after a blow. Pain 2 weeks.	..
56 5501	W. L.	21.7.98	Maria Kemp	L.	61	Sight failing 5 years. Pain 7 months.	..
57 5511	E. W. H. S.	6.8.98	William Dewsberry	L.	55	Eye defective and divergent for 20 years or more. Occasional pain for 4 years.	..

SARCOMA OF THE EYEBALL.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
live $\frac{11}{12}$ after excision.	Large mottled sarcoma with round and spindle cells. It extends from a point 7 mm. from O. D. on one side to within 3 mm. of O. S. on the other side.	T. +2, angle closed.	..	F. H. negative. No injury.
uite well 4 months after excision.	Large melanotic sarcoma with round and spindle cells growing by a pedicle from O. D. and reaching lens. Ciliary body not involved.	T. +2, angle closed.	..	F. H. negative.
live $\frac{6}{12}$ after excision.	Unpigmented mixed round and spindle cell sarcoma growing from equator at upper part.	T. normal, angle open.	..	F. H. negative.
live $\frac{5}{12}$ after excision.	Melanotic sarcoma, filling two-thirds of the subretinal space.	T. +1, angle closed.	..	F. H. negative. No injury or previous eye disease.
live $\frac{5}{12}$ after excision	Rather large sarcoma reaching from O. D. nearly to equator. Free from pigment. Small round cell growth. Broken down with hæmorrhage.	T. +3. No A. C. Angle closed.	..	F. H. negative. Sight failed after a blow 2 years before excision.
live $\frac{5}{12}$ after excision.	Small melanotic sarcoma near equator. Centre broken down with hæmorrhage. Much degeneration.	T. +2, angle closed.	..	F. H. negative. Detachment of retina was seen 5 years before excision.
live $\frac{4}{12}$ after excision.	Eye completely solid and filled with a dense black sarcoma. Centre is soft and broken down. Sclerotic has given way behind and growth projects into orbit.	..	..	F. H. negative. Contents of orbit removed.

Sarcoma of the Eyeball.

No. of case and No. in Hosp. Reg.	Surgeon.	Date of excision.	Name of patient.	Eye affected.	Age at date of excision.	Duration of previous symptoms.	Interval between excision and first symptom of recurrence.
58 5542	W. L.	8.9.98	Harry Goodchild	R.	50	Sight failing 3 years. Blind 1 year. Slight pain for 3 weeks.	..

SARCOMA OF THE EYEBALL.

Duration of life after excision.	Position, extent, and nature of tumour.	Tension and condition of angle of anterior chamber.	Site of recurrence.	Family history, remarks, &c.
live $\frac{3}{12}$ after excision.	Small mushroom-shaped melanotic sarcoma growing from posterior part of choroid. Retina completely detached.	T. full, angle closed.	..	F. H. negative. Slight injury 14 years before excision, but from this it quite recovered.

The Royal London

Ophthalmic Hospital Reports.

Vol. XV. Part II. May, 1902.

THE NERVE-SUPPLY OF THE LACRYMAL GLAND.

By J. Herbert Parsons, F.R.C.S.

It has generally been considered that the secretory nerve fibres to the lacrymal gland are derived from the fifth cranial nerve. Some observers, reasoning principally from the analogous salivary glands, have included the cervical sympathetic as an additional source of supply. More recently the view has been advanced that the true secretory nerve is the seventh and not the fifth.

The object of the present paper is to examine critically the evidence on the question from anatomical, histological, embryological, physiological, and pathological standpoints, to record some further observations, to point out some hitherto neglected methods of investigation, and to supply a bibliography of the most important papers upon the subject.

I. *Anatomy.*

The lacrymal nerve, derived from the first or ophthalmic division of the trigeminal, divides close to the gland into two branches. Of these, the upper is distributed in part directly to the gland, and partly passes out of the orbit to supply the skin of the upper eyelid, &c.; the lower branch communicates with the temporal branch (*ramus temporalis, subcutaneus malæ*, &c.) of the orbital or temporo-malar nerve,

which is derived from the second or maxillary division of the V, and is then distributed to the gland. Through this communication it is possible for fibres of the VII or of the nervus intermedius of Wrisberg to reach the gland, leaving the facial at the geniculate ganglion, and passing to the sphenopalatine (Meckel's) ganglion *viâ* the great superficial petrosal and Vidian nerves, and from the ganglion into the second division of the V, *viâ* the two spheno-palatine nerves.

That the communication with the orbital nerve is of some importance is shown by the occasional absence of the lacrymal, its place being taken by the orbital. On the other hand, the lacrymal has been known to supply the malar branch, which usually arises from the orbital. (Thane.)

It is thus obvious that purely anatomical considerations cannot settle the question.

II. *Histology.*

By means of the microscope the exact nature of the nerve fibres and their ultimate destination can be determined.

With regard to the latter point, Dogiel[1] has proved by the methylene-blue method that the fibrils are distributed in the same manner as in the case of the salivary glands. The nerve fibres which enter the gland are almost all non-medullated: they for the most part enter the lobules with the blood-vessels and ducts. They then form a plexus upon the basement-membrane of the alveoli. From this plexus fine branches pierce the basement-membrane, and form a network around the bases of the cells (Ueberzellennetz). Fine fibrils are given off from this network, penetrating between the epithelial cells, and there forming an intercellular network (Interzellennetz). Fibrils are sometimes seen which seem to end free, but Dogiel attributes this to incomplete staining. As in the case of other glands, it is doubtful if the fibrils ever actually enter the epithelial cells of the gland.

The cranial nerves have been especially the subject of histological investigation by Gaskell and his pupils. In the dog, the lacrymal is a branch of the second division of the V, and can thus receive fibres from the VII without the intervention of the subcutaneous malæ, with which it is said not to communicate. According to Edgeworth,[2] the lacrymal consists of "grey fibres and medullated fibres up to 16 μ in diameter." "The great superficial petrosal consists of grey fibres and medullated fibres up to 11·2 μ in diameter; it is a very small nerve, and contains but few fibres." It will thus be seen that it is impossible to demonstrate the passage of fibres of the VII or nervus intermedius into the lacrymal histologically, though the facts are not contradictory.

On the assumption that the lacrymal secretory fibres come from the great superficial petrosal, it is of interest to trace their origin from the brain. In the analogous case of the chorda tympani, which contains the secretory fibres for the submaxillary and sublingual glands, the fibres are derived from the nervus intermedius, which "is essentially a small fibred formation, with a few much larger fibres" (Gaskell[3]). The statement of Vulpian[4] that the chorda degenerates almost entirely after intracranial section of the V in rabbits, whilst it remains nearly intact after section of the VII in dogs, is opposed by the fact that the chorda contains far more medullated fibres than the great superficial petrosal, which is the only source from which it could receive fibres from the V in the tympanic part of its course. Moreover, according to the embryological researches of Dixon,[5] the great superficial petrosal grows out from the geniculate ganglion; and if this be true, the origin of its fibres, and therefore of the lacrymal secretory fibres, must be from the VII nucleus. On the other hand, Duval[6] claims to have traced the portio intermedia centrally to the upper end of the IX (glossopharyngeal) nucleus, in which case the origin of the chorda tympani, and probably also the lacrymal fibres, would be here.

III. *Embryology.*

The lacrymal gland arises from solid ingrowths of the conjunctival epithelium. The lacrymal duct is formed along the fissure which separates the lateral nasal process from the maxillary process, but its exact development is a matter of dispute; so that even if this be taken as a guide to the morphological relations of the gland—an assumption peculiarly open to criticism in the case of this duct—we are still lost in the unknown pre-mandibular region. Moreover, the morphology of the cranial nerves is so beset with difficulties and pitfalls that we can expect no aid from this source in solving the question of the innervation of the lacrymal gland.

IV. *Physiology.*

Historical Résumé.—Petit (1727)[7] found increase of tears after section of the "nerf intercostal" (cervical sympathetic) in the dog. Langley[8] attributes this to the hyperæmia of the gland due to paralysis of the vaso-constrictors, leading, as in the salivary glands, "to a greater secretion than normal whenever secretion is in any way set up." The fact has been confirmed by Arloing[9] in the deer and ox.

Czermak (1860)[10] obtained secretion from stimulating the V trunk, but in such a manner that it might be a reflex effect.

Herzenstein (1867)[11] obtained a positive effect from the lacrymal in sheep, dog, and rabbit, and a variable effect from the sympathetic.

Wolferz (1871)[12] confirmed Herzenstein's results, and also obtained an effect from the subcutaneous malæ. This can hardly be reconciled with the fact that the subcutaneous malæ does not communicate with the lacrymal in the domesticated animals.

Demtschenko (1871)[13], Tépliachine (1894)[14], and Tribondeau[15] also confirm Herzenstein, but the method used is open to the severest criticism. The outflow of tears was

apparently so small as only to be appreciated by pieces of filter-paper (Fliesspapier) placed in the conjunctival sac. The mere reflex effect induced by such irritation might account for the result.

Reich (1873)[16] performed 86 experiments upon rabbits, cats, dogs, and sheep in E. Brücke's laboratory. He found (1) that stimulation of the peripheral end of the V root causes no effect; (2) that reflex secretion can still occur after section of V intracranially; (3) that stimulation of the lacrymal nerve peripherally causes a flow, and that no reflex effect follows section of the lacrymal; (4) that stimulation of the cervical sympathetic causes a doubtful effect, whilst reflex effects may occur after section of V together with extirpation of the superior cervical ganglion; (5) that the secretory fibres do not come from the IV nerve; (6) that reflex flow may occur after tearing out the VII nerve. From (2) and (3) it is obvious that the lacrymal nerve in these animals contains secretory fibres which do not come from the V. In (6) the nerve was apparently torn out from the stylo-mastoid foramen, in which case the upper part, including the connection with the great superficial petrosal nerve, might very well have escaped injury. Reich's experiments are probably the most exhaustive and most careful hitherto recorded.

Vulpian and Journiac (1879)[17], in the rabbit, using curare (which is said to stimulate lacrymal secretion) and artificial respiration, stimulated the cavum tympani and obtained a flow of tears, and of a milky fluid at the inner canthus. The latter is derived from the Harderian gland. In two animals the whole of the intracranial part of the left VII was extirpated some days before, and much less result followed stimulation than on the right side.

Bechterew and Mislawski (1891)[18] found integrity of the V unnecessary for reflex effect. They affirm the "undoubted influence of the cervical sympathetic." They also obtained an outflow of tears from stimulation of the optic thalamus, which they regard as the reflex centre, and of the cortex,

especially in the neighbourhood of the sigmoid gyrus. They paid no attention to the VII nerve, and regard the V and the cervical sympathetic as the secretory nerves.

Laffay (1896),[19] as the result of intracranial section of the VII in the dog and repetition of the older experiments regards the VII as the secretory nerve.

Campos (1897)[20] describes at length a single experiment, in which he divided the great superficial petrosal in the monkey, and still obtained a diminished effect upon stimulating after time had elapsed for degeneration. From his description I should consider it doubtful whether he divided the nerve.

I have performed several experiments upon cats, dogs, and rabbits in which I have stimulated the lacrymal nerve, the subcutaneous malæ, the tympanic plexus, the VII intracranially, the V intracranially, and the cortex cerebri. In no case can one obtain a sufficient outflow of tears as to cause them to overflow on to the face. In over 20 experiments in which I have stimulated the cortex cerebri in cats for another purpose,[21] I have never obtained an undoubted secretion of tears.

Professor Schäfer[22] has shown that activity of the gland causes microscopic changes in the cells similar to those found in the serous salivary glands. These results, he tells me, were obtained by injecting pilocarpine. I have examined the gland after stimulating the lacrymal nerve for an hour and a-half, the dog being anæsthetised the whole time with A.C.E. mixture. The gland was hardened in Flemming's chromic-osmic mixture, and cut by freezing microtome. There were slight changes in the cells pointing to activity, but they were not marked.

The best result which I have obtained was in repeating Vulpian and Journiac's experiment in the rabbit. In the cat, I found that this experiment was accompanied by marked dilatation of the pupil, and it is noteworthy that Demtschenko obtained the milky Harderian gland secretion after stimulating the cervical sympathetic. Vulpian and

Journiac's degeneration experiments, however, seem to prove that the sympathetic cannot account for the whole effect. I have also found that stimulation of the cervical sympathetic is accompanied by blanching of the conjunctiva, whilst stimulation of the tympanic plexus usually gives vaso-dilator effects.

It is obvious, I think, that no conclusive result is likely to follow further research upon these lines. I would suggest the following experiments, which I hope to carry out at a future date :—

(1.) Intracranial section of V and examination of the lacrymal nerve, and the gland by the methylene-blue method after time has been allowed for degeneration.

(2.) Intracranial section of VII and similar examination of the lacrymal nerve and gland. Part of the nerve should be teased, and the remainder stained with osmic, and cut in paraffin, and compared with the intact nerve of the opposite side.

(3.) Examination of the electrical variations of the gland on stimulation of the various nerves mentioned, and comparison of the results with those obtained by Bayliss and Bradford[23] in the case of the salivary glands.

(4.) Further microscopic examination of the cells after prolonged stimulation of the nerves electrically.

V. *Pathology.*

Paralysis of VII.—Jonathan Hutchinson (1876),[24] "Paralysis of the Portio Dura of the right side, with deafness occurring suddenly. Paralysis of lacrymal gland, and temporary paralysis of lower extremity on same side. Slight defect of sensation on same side of face. No pain."

The patient was a woman, æt. 34. The paralysis came on two days after confinement.

Goldzieher (1876),[25] woman, 26. R. eye, episcleritis with iritis. L. eye normal. Complete paralysis of L. VII with lagophthalmos. Skin of L. side of face hyperæsthetic after

electrical treatment. Syphilis excluded. Woman wept whilst under observation, and frequently afterwards. L. eye remained quite dry. Tears returned on L. side when the paralysis passed off, and have since been quite normal.

Uhthoff (1885),[26] woman, 25. R. trigeminal neuralgia and paraesthesia of second division of V. Tears only from L. eye when she wept, which she frequently did with the pain. Diagnosis: neuritis, with paresis of VII fibres in maxillary nerve.

Goldzieher (1894), woman, 21. Congenital complete paralysis of L. VII with lagophthalmos. Sensation of skin of face and bulbus normal. Tears only on normal side since birth.

Jendrássik (1894)[27] (1) woman, 43. Complete paralysis of L. VII with lagophthalmos. No loss of sensation. Paralysis of lacrymal gland on L. side.

(2) Woman, 54. Complete paralysis of L. VII and of palate. Sensation normal. Less reddening of conjunctiva, and no tears. Recovered with recovery of facial paralysis.

(3) Woman, 44. Similar to (2).

(4) Woman. Incomplete paralysis of L. VII coming on 15 days after confinement. There were intermittent spasms, accompanied by flow of tears from L. eye only.

Jendrássik gives a very careful and minute account of the cases, but attaches too great importance to the paralysis of the palate considering our knowledge of its innervation.

Irritation of VII.—H. Schüssler (1879),[28] Actor's Cramp. Stretching of VII. Recovery. During violent stretching of VII an eggspoonful of tears gushed out of the eye on the same side.

Lumniczer,[29] another similar case.

Extirpation of Gasserian Ganglion.—Fedor Krause (1893).[30] After the operation neither more nor fewer tears flowed on one side than the other.

Horsley (1901)[31] writes: "The only point *re* lacrymation is that the hyperlacrymation ceases. I confess I have not examined the mucous membrane carefully to see whether

there is any *defect* of secretion. No patient has made any complaint about this." This is of great importance, considering Professor Horsley's wide experience of these cases.

It should be pointed out that complete paralysis of the VII is comparatively rare, the lesion in the majority of cases being at the stylomastoid foramen. In these cases the great superficial petrosal is not involved, and we should expect the secretion of tears to be normal. Indeed, many cases of lagophthalmos are accompanied by Stillicidium lacrymarum, the outflow of tears being particularly evident owing to the immobility of the lower lid. We must further bear in mind the possibility of a paralytic secretion analogous to that described by Langley for the salivary glands.

In conclusion, while the question cannot be considered settled, there is yet a sufficient amount of evidence in favour of the facial nerve being the secretory nerve of the lacrymal gland to cast much doubt upon the generally received view in favour of the trigeminal.

[The experiments were performed in the Physiological Laboratory, University College, London.]

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A CASE OF SYMPATHETIC INFLAMMATION AFFECTING THE POSTERIOR PART OF THE UVEAL TRACT.

By J. Herbert Fisher, M.B., B.S. (Lond.), F.R.C.S., Assistant Surgeon to the Hospital, Assistant Ophthalmic Surgeon to St. Thomas's Hospital, &c.

Of all the varieties of eye disease which have from time to time been classed as sympathetic, we may, I think, at the present day recognise two main classes. With the larger class, sympathetic iridocyclitis, every ophthalmic surgeon is, alas! too familiar; the plastic character of the inflammatory process, its intractability and often disastrous results, are well known to all; instead of the more usual terms, some authors refer to such cases as sympathetic irido-choroiditis. The other class forms quite a small proportion of the total number of recorded cases of sympathetic disease. The examples of this smaller class, as far as I have been able to find them, are invariably called cases of neuro-retinitis or papillo-retinitis. In the exhaustive and very valuable Report of the Committee of the Ophthalmological Society of the United Kingdom, published in the Transactions, 1886, vol. vi, eight or nine cases of this variety of sympathetic inflammation are reported, all under one or other of these designations. The case by Colsmann, there included and originally published in the Berlin Klin. Wochenschr., 1877, the Committee regarded as doubtfully of sympathetic origin, as it took the form of neuro-retinitis only, and they had doubts also about one or two others. The prognosis in these cases was found to be far better than in those belonging to the larger class; nearly all recovered quite good vision, and the observation is made that this favourable result seems to occur whatever the interval between the dates of injury to the exciter and onset in the sympathiser may have been. I have been able to find reports of a few other cases; the recovery of vision in nearly all of them seems to be quite remarkable. I find them invariably entitled cases of neuro-

retinitis or papillo-retinitis by their authors; they are never referred to as cases of sympathetic uveitis or choroiditis. In some, however, definite choroidal change is described by the reporting surgeon, though he regards this as of subsidiary importance, *e.g.*, V. Graefe (Arch. für Ophthalmol., xii, ii, 171). The left eye was operated on for extraction of a calcified lens; six weeks later rapid painless failure of sight occurred in the right eye, due to diffuse retinitis; this was followed by keratitis punctata and paralysis of accommodation. Two and a-half months from the onset the vision had improved to $\frac{4}{5}$; disseminated spots of choroidal disease appeared towards the end of the case. A case reported by Krause (Hirschberg Centralbl., 1881, p. 7) seems to belong to the common variety of sympathetic disease, but choroiditis patches were noted in the lower part of the fundus of the sympathising eye. Galezowski (Traité d'Ophthalmoscopie, 1886, p. 202) reports two cases that he had under care, and calls them cases of sympathetic lesion of the retina; in each, however, he says the choroid had undergone at the same time change in various places in the shape of obvious disseminated atrophic patches. Such spots of choroidal atrophy he was inclined to think were due to pressure of blood, and from the context it was the blood poured out in the retina to which he referred. Such a cause for the formation of spots of atrophy in the choroid coat is new to me, and I must confess does not seem to me very probable. I am not aware that spots of retinal hæmorrhage from other causes on clearing are in the habit of revealing areas of atrophied choroid. This then is the class of case on which those who argue for the transmission of the existing cause of sympathetic inflammation by the route of the optic nerves and chiasma rely, while cases of the more ordinary variety occasionally also present changes at the disc. The Committee of the Ophthalmological Society came to the conclusion, however, that the fact of papillitis showing itself did not prove transmission by the optic nerves, as it was often present in simple cases of serous iritis. The rarity of papilliitis in cases of

sympathetic inflammation is a point advanced by the opponents of Deutschmann's theory, but with the conflicting views I do not propose to deal.

When the case which I here report came under my care I was not so well acquainted with the literature of this variety of sympathetic disease as I have since been able to make myself, and I was in the habit of referring to my case as one of sympathetic uveitis, in which the most posterior segment of the uveal tract was bearing the brunt of the disease. This seemed to me to be the natural view to take, and I must say what I have since learnt has not as yet led me to acquiesce in the more general view. It seems to me improbable that at one time the sympathetic influence shall direct all its energy to the iris and ciliary region, while at another time it will leave the uveal tract severely alone and select the retina and disc for its operations.

All the cases, including my own, have in the active stage and over a long period been accompanied by a quite alarming reduction of vision, while the recovery of function has not been less remarkable. In my own case the vision was reduced to the ability to count fingers at 2 or 3 feet, and that only by eccentric fixation, and the patient was under my care and observation for two months before any definite improvement in the sight could be detected, though repeatedly tested. During all this period, and for long after, there was very active pathological fundus change. I doubt if any retina or optic nerve could be in so active a condition of inflammation for many months and the eye eventually recover full direct vision and a full field as my case did, with no sign of atrophy at the optic papilla. Another point which much interested me, and to which I think much weight may be attached, is the fact that there was very definite reduction of tension in the affected eye; this is a point about which there was no doubt whatever; observations were repeatedly made and recorded by the house surgeons and myself. On admission the tension of the sympathising eye was normal; soon it became reduced;

a fortnight after onset it was −1; very gradually it improved. I was in the habit of saying to students that I considered this fall in tension as showing that the vascular uveal tract was performing its nutritive duties imperfectly to the vitreous and aqueous humours of the eye as well as to the retina. As the result of the softening of the eye, I pointed out that the globe was squared by pressure of the recti muscles, and when the tension began to show signs of improvement I held this to be the first good sign, and was hopeful that before long the retina also would be better treated by its nourishing coat and show some improvement in appearance and recovery of function, and this subsequently took place. I regarded the choroid as being in a state of diffuse inflammation, and the changes in the retina and at the optic disc I think can be sufficiently accounted for as œdema secondary to the disturbance of its nourishing tunic. If it is argued that in the end the choroid showed no change on ophthalmoscopic examination, the argument applies, I think, with still greater force to the retina if any one is inclined to say that retinitis was the main feature. I conceive it is possible for such a coat as the choroid to be diffusely inflamed and to be left generally somewhat sclerosed without the change being conspicuous on ophthalmoscopic examination. In this condition the chorio-capillaris might still retain a sufficiently free circulation to allow the retina which it helps to nourish to regain its function to the full. A severe primary retinitis is rarely so devoid of retinal hæmorrhage as mine was.* It is not likely to cause reduction in tension in the eye, to continue for many months with com-

* My patient showed one very minute streak of retinal hæmorrhage, found towards the end of the case. Galezowski's patients appear to be two in whom retinal hæmorrhages were present; in none of the others to which I have been able to refer is any mention made of blood extravasation in the retina. It is only to be expected that with turgid retinal veins from obstruction by a swollen optic disc hæmorrhage would occasionally be seen; it is hardly to be believed that so many cases can be accurately described by the term sympathetic retinitis, when in the great majority no retinal hæmorrhages can be found; distended veins are observed in nearly all of them.

plete recovery of vision, and to leave practically no ophthalmoscopic change.

No one will, I think, deny the sympathetic character of the disease. The boy had always had perfect health; there was nothing in his appearance to suggest congenital syphilis; his father, mother, and eight other children were all living and enjoyed good health; the patient was carefully examined while in hospital and all organs found to be healthy. The most anterior part of the uveal tract did not quite escape; the iris at first was muddy; two posterior synechiæ had formed, but gave way gradually to mydriatic till the pupil was quite round: there was slight keratitis punctata only, but a punctate deposit of brownish and grease-like spots, identical with those seen on the back of the cornea, was abundantly present on the anterior lens capsule, and frequently examined with the pocket magnifier; there was also fine vitreous opacity.

H. B., æt. 18, baker, was sent to St. Thomas's Hospital by his doctor on January 9, 1899, with the following history:— Six weeks before admission, when attempting to find his way out of a dairy in the dark, he struck the left eye against the latch of a door. The same evening he went to his doctor and remained under his care. By Christmas week the injured eye had become quiet, and he returned to work. On December 27, 1898, he was taken ill with pains in the head, and also expectorated blood, mixed with phlegm. His doctor again saw him the next day and diagnosed influenza, but the symptoms do not appear to have been very marked. At the onset of the influenza the left eye again became inflamed, and remained so until I saw him; its vision did not recover at all after the accident. The right eye first became inflamed five days before admission to hospital; its vision began to fail at the same time, and continued to get worse. Belladonna lotion had been used to the left eye throughout, and to the right also, from the time it began to show symptoms.

To his family and personal histories I have already alluded; it is sufficient here to say that both are perfectly good and against any form of constitutional inheritance.

On admission to hospital the right eye showed conjunctival and slight ciliary injection. Cornea clear. Iris rather muddy. Pupil moderately dilated. Tension normal. V. = counts fingers at 1 metre. Oph: O. D. muddy, and edges quite obscured; general œdema of retina, but no cherry-red spot at macula; veins very much swollen; no material alteration in size of arteries; no hæmorrhages. Left eye: conjunctival and ciliary congestion. Scar at upper margin of cornea and extending in sclera. Iris above tucked back, simulating a coloboma. Iris muddy. Pupil moderately dilated. Some opacity of lens. No fundus reflex. T. −. Shrinking pain on pressure in ciliary region. V. = hand shadows.

The urine was normal, and all thoracic and abdominal viscera healthy. Mr. Lawford kindly saw the patient with me, and we agreed that the case was one of sympathetic disease of the right eye, and that the left should be excised. I accordingly removed the injured eye three days after admission; in the meantime no material change had occurred in the right. He was taking small doses of Hydrarg. c̄ cretâ, and atropine was being used.

On January 16 I found the right eye rather less congested, but it flushed on exposure to light. The cornea was clear. The pupil now well dilated and circular; the iris brighter; two small posterior synechiæ were obvious at the temporal part of the papillary margin. T. = −1; V. = fingers at 5 feet.

January 18. I noted fine dotted deposit on the anterior lens capsule, none on the back of the cornea; a little, but not much, fine opacity floats in the vitreous. Fundus now is a good deal more obscured; reflex rather greyish-red. Optic disc and retina give the same impression as before, but are by no means so clearly seen.

January 27. Pupil now wide and circular. T. still −. No pain. Abundant dotted deposit quite like that of keratitis punctata, but situated only on the anterior lens capsule, and chiefly on its inner sector; fine deposit in vitreous. Vision not appreciably improved. Fundus dimly visible; no outline of optic disc can be seen; distended tortuous veins everywhere; no localised choroidal exudation discoverable, but a general infiltration of this coat is suggested by the ophthalmoscopic appearances. I ordered two leeches to the right mastoid region.

On February 1 I stopped the Hyd. c̄ cretâ which he had been taking, and substituted a mixture of Pot. iod., with salicylate of soda and liquor ammonii acetatis, and ordered him a subcutaneous injection of nitrate of pilocarpine every other night. The pilocarpine was used in doses of gr. $\frac{1}{10}$th, and the dose never needed to be increased; he tolerated it very well; it produced profuse diaphoresis, and was continued until March 2. After this date small doses of Donovan's solution were prescribed; atropine was of course used throughout.

On February 8 I made the following note: "Still much dotted opacity on anterior surface of lens; none made out on back of cornea. Vitreous is clearer, but still shows floating opacity; some of this is cobwebby and larger than I have seen in this patient before. Fundus is much more clearly seen than at last note. Edges of optic disc quite lost; slight swelling of papilla, I think about 2·5 D. Veins turgid; in peripheral parts of retina are to be made out small circular dead-white dots, not glistening; the whole retina is hazy and œdematous; no hæmorrhages. V. = counts fingers at 6 or 8 feet; this he does by eccentric fixation, turning the eye up to count fingers held on my coat. T. is improving, but is still slightly reduced. He finds his way about readily, *e.g.*, in and out of the dark room, and collides with nothing. He stands the pilocarpine well, and the $\frac{1}{10}$th gr. has not yet had to be increased."

The next extract is my note of February 22, which reads as follows:—

"Two or three small grey dots of keratitis punctata on back of cornea; the pigmented dots on anterior lens capsule are less numerous. Cobwebby vitreous opacity still renders the whole fundus hazy; retina and optic disc are both œdematous, the former especially in its lower part. No hæmorrhages seen. I find less of the white dots in the fundus to-day than a fortnight ago; some are still, however, present; one, in the nasal half of the fundus, is crossed by one of the inferior nasal vessels. The eye is practically quite quiet, not flushed or photophobic, and its tension is certainly improved—indeed, very little below normal."

On February 27 the house surgeon recorded again his observation of keratitis punctata and noted, "tension normal," to which I agreed. From this date the tension kept normal, but

the globe remained squared by pressure of the recti muscles for some months. On March 2 the patient thought vision was better; said he saw better in twilight; still fixed eccentrically.

The next note I will quote is that of March 15. "Will count fingers at nearly 6 yards to-day, but not read any letter on Snellen's types. No longer any keratitis punctata to be made out, and on the anterior lens capsule there are only two or three dots of brown iris pigment, and no deposit from the ciliary region. Vitreous haze has practically all cleared, and fundus details are in every way much more distinct. Œdema of lower half of retina has nearly vanished; to outer side of inferior vein I see a group of circular exudation spots; the others found mostly in nasal half remain unpigmented and brightly glistening; optic disc is still swollen 3·0 D.; its outline streaked; no hæmorrhages. Vessels of good size; physiological cup remains a very obvious funnel. Tension normal."

On March 22 I observed a small area of mottled change at the macula and a suggestion of redness like that seen in embolus cases in the acute stage. A week later I found one quite short streak of hæmorrhage in the retina about one disc's breadth above the yellow spot. On April 5 the spot of hæmorrhage was still present, and vision, under atropine, with + 0·75 D. sph. = 6/36, one letter of 6/24, and this seemingly by central fixation.

On April 8 I discharged him from the ward, wearing goggles, and taking with him atropine drops.

On May 3, in the out-patient room, the eye with + 1·0 D. sph. saw 6/12 partly. The disc was still slightly swollen. I need not quote the full ophthalmoscopic note. I put him on quinine and iron.

On May 31 V. was 6/12 with + 0·75 D. sph., not 6/9. The swelling of optic disc was nearly subsided; the outline and surface brighter and sharper, but the former still radiate and softened. A good deal of patchy pigment disturbance near the macula and between this and the disc. The white circular dots found chiefly in the nasal portion of the fundus show slight pigmentation, and one at least is distinctly behind a retinal vessel. Goggles and mydriatic were abandoned.

September 6. "Says the sight has got much clearer. Is at work, driving a pony and trap. Vision to-day is 6/6 in a dull

illumination. Field for white taken on the perimeter quite full. Optic disc normal. Some white spots apparently going to remain permanently in the upper and nasal parts of the retina; the macular region shows so speckled an appearance one would not expect the eye to see 6/6ths, as it does. Tension normal."

The following report on the condition of the exciting eye was furnished me by Mr. Devereux Marshall, and my best thanks are due to him for his very careful examination :—

"There appears to be a coloboma upwards. Eye opened in an equatorial direction. There is some infiltration of vitreous, starting from a point where there has been a wound. The retina is wrinkled, and on one side, the lower, there is extensive retinal detachment, beneath which there is much coagulated albuminous material. Optic disc somewhat swollen and indistinct.

"*Microscopical Examination.* — The wound at the corneo-scleral junction is plainly visible, and apparently the ciliary body has been carried away from its attachment and has allowed the iris to be turned backwards between it and the sclera. Although the iris cannot definitely be seen it is surmised the above must be the explanation of the coloboma-like appearance, as some pigment lies between the sclera and the ciliary body, which is probably iritic in origin. The angle on the opposite side is closed, and the iris is thickened, inflamed, and its vessels enlarged. The ciliary body is intensely inflamed, so also is the choroid and retina. The lens is *in situ* and but little affected, though it is drawn somewhat out of shape near the ruptured sclera. The back part of the eye shows very marked changes. The choroid is about six times its normal thickness, and composed of masses of small round cells with large vessels; much pigment scattered through it. At one spot near disc the retina and choroid are firmly adherent. The optic disc is somewhat swollen, and there are collections of small round cells in it. The substance of the nerve does not seem to be affected. Cross-sections of the nerve show some inflammatory changes about the sheath, but the nerve is unaffected, *i.e.*, there is no proliferation of nuclei, &c. The fibres are indistinct and appear swollen. Vessels are much enlarged and distended with blood. Numerous small vessels run through the nerve in all directions."

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CASES AND OBSERVATIONS,

By E. Nettleship.

I. On Attacks of Temporary Blindness from Exposure to Cold Wind.

Case 1.—The Rev. —— (26.123) came to me in November, 1892, when æt. 43, about reading-spectacles, and gave the following history:—He usually spent his August holiday in Switzerland, often ascending some of the high passes. The first time he did any high climbing, about 6 years ago, when going in the teeth of a cold wind over a pass 10,000—12,000 feet (3,000—4,000 metres) high in perfectly clear weather, he became rapidly almost blind of both eyes for some time. It was like an extremely thick white fog; he was unable to see his feet, and the guides had literally to place his feet for him, or he would have missed his steps. As soon as the party were over the pass they were protected from the wind; his sight then began to improve, and was soon restored perfectly.

An exactly similar attack occurred another year in similar circumstances, but this time vision did not become quite clear till he had had a night's rest.

Again in August, 1894, in ascending the Rothhorn against a cold wind, he suffered for a third time in the same way. I had asked him to notice particularly how the defect of vision began, and he told me that on this occasion he noticed first broad rainbow fringes on each side of the rope, and next a mistiness between him and the ground, so that he began to think he should again lose sight of his feet. The attack came on shortly before they reached a certain rock shelter, which, though near the top, was protected from the wind. The symptoms passed off on reaching this shelter, and did not return when he descended the other side, "but the wind was not then in our faces." He was wearing goggles, as he always does when mountaineering, and had also taken the precaution to protect his head with a thick woollen wrap. The attack came on about 8 o'clock A.M., between the first breakfast and the second. He

had been at the Riffel Alp Hotel for a day or two before making this ascent, and it was there that, on the following day, he gave me the account of this last attack; he had been in Switzerland, lower down, for a fortnight before. He told me he had never in any of his climbs been troubled by giddiness, faintness, or difficulty of breathing at high altitudes.

The attacks never happen except when facing a cold wind on the Alps, and he is convinced that the loss of sight is the result of the cold wind upon his eyes. It was not the effect of snow dazzling, for he always used goggles, and, moreover, on the last occasion (1894) he was on dark rock free from snow when the dimness came on, though snow had been traversed lower down.

He has always been "a chilly subject," likes to wrap up warmly, is spare, with congested cheeks and nose, and very cold feet, is liable to chilblains on the ears (though not on hands or feet), but never has "dead fingers." He is subject to hay fever. He has the sort of ocular irritability that is shown by dislike of separate lights in a room, and always reads with a shaded lamp; but diffuse daylight, however strong, does not annoy him. He uses the microscope for studying minerals, and believes that polarised light tires his eyes more than ordinary light. Though, as stated, he has never had a real attack of dim vision except on the high Alps, he has several times had slight appearances of the same mistiness when facing cold wind even in England. There is no history of rainbows or evening mists suggestive of glaucoma. He has never had snow conjunctivitis.

I found he had full V. with slight H. As., and required

+ 0·75 D. s. + 0·5 D. c. for reading.

In each eye the lower retinal vein crossed its companion artery at the disc and showed spontaneous pulsation, there being no pulsation in other veins, which did not cross arteries. In the R. retina below O. D. were a considerable number of Gunn's dots ("Crick dots" of Mr. Marcus Gunn),* but I could see none in the L. He counted his R. as slightly the better eye for microscopical and other accurate work.

* Marcus Gunn: "Peculiar Appearance in the Vicinity of the Optic Disc occurring in Several Members of the same Family," Transactions of Ophthalmological Society, vol. iii, p. 110, 1882.

Case 2.—Arthur C., 25, told me in February, 1900, that for some two or three years he had been subject to attacks of "blindness" in his R. eye, lasting from half an hour to an hour, less or more. The attacks never came on except when he was driving in cold weather combined with a high wind (his work is to drive a tradesman's cart in a very exposed district 800 feet above sea-level). The appearance was as of a thick white mist before the eye, not actual darkness; it was so dense that if he shut the other eye he could see no objects at all with the affected one. He prefers to shut the affected eye when the attack comes on, but asserts repeatedly that "the dimness is there just the same whenever I open it." There is no pain or inflammation. When he gets into the house and gets warm the sight returns to its ordinary state.

The eye in question (R.) is not quite so good as the other owing to an old nebula of cornea running downwards from the level of the lower border of the pupil, which has the aspect of old interstitial keratitis. The L. cornea is normal. Fundus pupil and tension quite normal in both eyes. His physiognomy was suggestive of inherited syphilis. He could give no account of previous inflammation of the R. eye in explanation of the nebula.

It is probable that the keratitis was associated with a permanent change in the arterial coats of the choroid and retina of this eye, which, though not noticeable to ophthalmoscopic inspection, impaired their elasticity as compared with the vessels of the other (healthy) eye.

In the above cases the failure of sight had not the characters of the temporary blindness sometimes seen in persons with cardiac disease, and believed to be the expression of obstruction—as temporary as the loss of sight—in the arteria centralis by embolism or by thrombosis, perhaps aided by embarrassment of the blood-stream due to valvular mischief. The phenomena are much more suggestive of partial arrest of the retinal or choroidal blood supply by the paralysing effect of cold in a person whose terminal circulation, as in the subject of the first case, was very feeble. The question is whether cold applied to the surface can cause contraction

of the deeply lying vessels? I believe that few would have accepted such an explanation—I certainly should not—even tentatively, until the publication by Hertel* of the results of his researches on the effect of heat and cold applied to the surface of the eyelids. According to Hertel the temperature not only of the conjunctiva, but of the tissues of the orbit behind the eyeball—and presumably, therefore, of the structures of the eyeball itself—is lowered several degrees by cold and raised several degrees by heat applied to the surface. This effect he attributes mainly to conduction through the tissues, not to deprivation of blood by contraction of arteries, though the range of alteration of temperature appears to be increased if the circulation be stopped.

Assuming the correctness of Hertel's conclusions, we can interpret the cases above given by supposing that the cooling by conduction caused marked contraction of unusually susceptible retinal and choroidal arteries, and that the anæmia produced was sufficient, either alone or in combination with the lowered temperature, to depress almost to extinction the function of the retina.

It seems very desirable that Hertel's experiments should be repeated; the questions involved are of great importance.

* Hertel: "The Influence of Cold and Warm Compresses on the Temperature of the Eye," Graefe's Arch., xlix; abstract in Ophthalmic Review, vol. xix (1900), p. 18.

II. Berry's "Hyperplastic Subconjunctivitis" and Allied Conditions.

In 1892, Dr. G. A. Berry described at the Ophthalmological Society (Transactions, vol. xiii, 219) a peculiar condition of hard swelling of the parts beneath the conjunctival fold of the lower lid, under the title of "hyperplastic subconjunctivitis," and about the same time he described and figured the disease in the 2nd edition of his text-book, p. 113. He states that the condition is accompanied by little or no conjunctivitis or irritation, and that it disappears after some months without treatment, but adds that there may be chemosis, and swelling of the pre-auricular and even of the more distant glands.

The first of the cases given below shows that the condition described by Mr. Berry may sometimes be of serious importance as an early sign of Hodgkin's disease, and suggests that the glandular enlargements mentioned by Berry as having sometimes occurred in his cases may have had more than a passing importance.

Case 1.—Miss N., æt. 56 (P. 23.105) was first seen in July, 1891, with a peculiar semi-solid thickening of the entire retrotarsal part of the conjunctiva of each lower lid; the thickened part was whitish and slightly translucent, and in the affected part of the L. lid, which was rather worse than the R., a few minute punctate hæmorrhages could be seen; there was only insignificant congestion. The upper lids were quite natural. She said that the cheeks had also lately been slightly swollen and stiff, but that it was "not erysipelas." V. 6/12. Slight myopia. Unfortunately I did not examine for enlarged glands.

The affection of the lower lids had begun several months before, when she was at Bournemouth on account of a tendency to bronchitis. She said the amount of irritation of the eyelids varied from time to time, but had never been worse than at present.

I neither saw nor heard any more of Miss N. till March, 1892, when Dr. Bramley Taylor, of Denmark Hill, asked me to see her with him at her house. She had been ill ever since the end of the previous summer, when Dr. Taylor found her urine to be very albuminous and containing some blood, but at no time could he find casts; during the winter she had some bronchitis and had scarcely been out of the house. There had been no œdema or swelling anywhere except in the lids and face. She had lost much flesh and become very pale. I understood that neither Dr. Pavy, who had seen her, nor Dr. Taylor had been able to detect enlargement of any glands except those below the jaw (about to be mentioned), nor any enlargement of spleen. The blood had not been examined, but I believe an examination was made within the next few days. At the date of our consultation the condition was as follows:—The swelling of the conjunctiva, though of exactly the same character as in July, 1891, had much increased and now affected both lids of both eyes, but was still limited to the retro-tarsal part of the conjunctiva, not passing back into the orbits at all and only invading the overlying skin to a slight degree; there were now no ecchymoses. On L. side the swelling was so great that the lids were almost closed and could scarcely be opened even by traction with the fingers; the lids however were very easily everted, and then, owing to the roll of dense tissue, were difficult to replace; there was no proptosis and the ocular movements were quite free. Eyes watery but free from sticky discharge. Sight not affected. Fundi normal;—no hæmorrhages.

About the root of the nose and in a less degree in the cheeks there was brawny, mostly subcutaneous, œdema, and in the submaxillary region on both sides were a number of loose, bullety glands. These were of no great size, but there must have been more deeply seated thickening also, for there was an impediment at the articulation of the lower jaw that prevented the mouth from being opened more than half an inch; this had come on recently and was increasing. No enlarged glands could be found anywhere else, nor was there any œdema, except in the face; her neck, body, and limbs were extremely thin, contrasting strongly with the face. I did not see her again, but Dr. Taylor told me that the cervical glands became much enlarged and that

the patient died rather suddenly about a fortnight after the above date, having for the last three days of her life suffered much pain in the shoulders and arms.

In the following case the same part of the palpebral conjunctiva was affected and apparently in a very similar manner, but only for a short time; and, in the eye first affected, the conjunctival swelling accompanied an attack of subacute iritis.

Case 2.—Jane W. (G. B. 2.14) came to St. Thomas's Hospital on February 20, 1891, for iritis in the R., with deposit of white lymph at the lower part of a.c., and numerous adhesions; the vitreous and fundus were normal; keratitis punctata not mentioned; the eye had been affected for a fortnight and there had been no severe symptoms. The only peculiar condition was a "semi-solid milky infiltration of the posterior part of the conjunctiva of the lower lid, covered with a thin muco-puriform pellicle." She was rather pale, had a chronic cough, and menstruation was excessive; no history of syphilitic symptoms. No enlarged glands detected. Atropine, bandage, and iron with quinine were ordered, and within two or three days the retrotarsal œdema had quite disappeared though the lymph was still present in a.c. But now (February 24) an exactly similar state had developed in the L. lower lid, though atropine proved that there was no iritis in this eye; by 27th this lid also was well again, and an examination of both eyes again showed the absence of disease of the vitreous.

It seems probable that this case began with iritis and that, owing to the state of the patient's health or of her blood, the ocular inflammation caused temporary deep lymphatic congestion or obstruction which afterwards spread to the opposite side.

In the next two cases a very chronic semi-solid œdema occurred in the lower part of the ocular conjunctiva, leaving the eyelid unaffected. The condition was limited throughout to one eye. No cause could be found, but it seems almost certain that some undetected local obstruction in the lymphatics of the neighbourhood was present.

Case 3.—Mrs. B., æt. 62 (24.226) was sent by the late Dr. Richard Gravely, of Newick, on March 22, 1892, with a very marked degree of semi-solid œdema of the lower part of the ocular conjunctiva of the R. eye; it had begun in the previous January without apparent cause; there had been no pain and only trifling irritation. The L. was quite natural. A month later there was no alteration, though she had used a lead-lotion compress to the eyelids with more or less regularity. Dr. Gravely could find nothing wrong with the teeth or antrum. The eyes were widely different in refraction, the R. H.m. 1 D. V. 6/9, the L. (used for reading) M. 5 D. V. 6/12; but both were healthy in all parts so far as ophthalmoscopic examination showed.

She said that on two occasions she had had "a red streak of lumps on the R. forehead about a finger's breadth," lasting perhaps a fortnight, but not itching nor weeping; also that once or twice she had had a moist eruption on the cheeks and forehead. She was subject to "rheumatic stiffness" of the knees, but without swelling. I did not see Mrs. B. again, but Dr. Frank Gravely writes in answer to my recent inquiry (Dec. 1901) that soon after I saw the patient in 1892, she had a sharp attack of inflammation in both eyes (caught in visiting at a cottage, the patient thinks), that when this subsided the œdema of the R. conjunctiva was found to have disappeared, and that the eyes have remained quite well ever since.

Case 4.—Charles P. 61 (G. B. 2.37), a chair-maker, living at Battersea, came under my care at St. Thomas's Hospital, in June, 1892, with "semi-solid transparent œdema of the lower half of the ocular conjunctiva" of the R. eye extending quite up to the corresponding part of the edge of the cornea: I particularly noted that there was no infiltration of the retro-tarsal part. A week later slight quiet œdema of the corresponding cheek and external aspect of the lower lid was noted; it may have been present when he first came. He had lost all his upper teeth, and there were no signs of disease of bone or antrum. The urine, examined more than once, contained neither albumen nor sugar. He had somewhat failed in strength the last two years, but had had no illness. When a child he had small-pox, and

some pits were still visible. No enlarged glands could be found. The nose was not examined.

He said the eye had been like this for 8 months before I saw him, and he remained under my care for 8 months (till February, 1893), making, say, a year and a-half in all, and even then the condition, though much less marked, was not cured. He said that it began without any severe inflammation, and certainly no conjunctivitis occurred whilst under our notice, though he complained at times of smarting. He said the swelling was rather less every morning and got worse as the day passed; and that it was always made worse by hot air and relieved by cold.

He used various lotions and kept the eye tied up as much as he could—at one period for as much as 4 days a week, and every night—and took iron and arsenic. After 7 months of such treatment as an out-patient the condition was (January 17, 1893) neither better nor worse. At the end of January, I took him into the ward and kept him in bed continuously, with the eye bandaged, for a week, with the result that the œdema entirely disappeared; but on February 14, when he had been up again for some days, it was beginning to return, chiefly on the nasal side, in spite of the bandage being applied every morning before he left his bed and worn all day till bedtime. He does not seem to have come again after this date.

III. On some Cases of Partial Albinism.

It is well known that albinos suffer almost always from nystagmus and defective acuteness of sight; that there are many degrees of albinism; that in some albinos gradual pigmentation of the hair and skin takes place, so that in after life the term, in so far as it is based on the complexion, may cease to be applicable; that a considerable amount of ametropia is a common accompaniment;* and that the condition often affects more than one in a family.

We also meet with cases of nystagmus with unexplained imperfection of sight, ametropia and blue eyes, *i.e.*, incomplete pigmentation of irides, occurring in more than one of a family. In one such instance that I recorded in vol. xi, p. 366, of these Reports, nystagmus occurred in no less than six males of the same stock; all had blue eyes, and the two of them whom I saw had a high degree of astigmatism, chiefly hypermetropic. It was stated that no cases had occurred in females, and none in males with brown eyes, and the one brown-eyed boy that I saw proved to be emmetropic with steady eyes. The fundus in one of the affected boys was "pale in the y. s. and o. d. region, but there was nothing unusual, and certainly nothing positively unnatural" in the appearances. These six affected boys belonged to three families of the same stock, and the inheritance in all was through the mother.

In 1882 Mr. Lloyd Owen published a remarkable example of nystagmus affecting many males of the same stock through no less than four generations, and here again transmission was always through the mother, though no females whatever were affected. In several of the children in this series the

* M., according to Fuchs; As. and other forms of Am., according to Landolt; H., according to Hutchinson in these Reports, vol. vii, p. 37. I have notes of refraction in 14 cases (including those published or referred to in the present paper)—there was considerable H. or H. As. in 9, almost Em. in 3, high M. in 2 (both children).

hair was fair, and the fundus oculi of light colour from subnormal pigmentation; but several of those affected in the previous generation, and therefore much older at the time of publication, had dark hair. The vision, refraction, and colour of the eyes is mentioned in only one of the whole series, a boy of 15 who had blue irides, light brown hair, and very light coloured choroids, and was hypermetropic, with very imperfect vision; but we may take it as probable that some if not all of the other affected individuals also had blue eyes, and it would seem that all of them were more or less amblyopic.

Mr. Lloyd Owen's paper* was entitled "Hereditary Nystagmus," but partial albinism seems to have been present in his mind, for he says that the fundus in one of his cases was "lighter than is normal, even in light-haired people short of albinism." My own case, above referred to, was recorded as illustrating one amongst several forms of congenital amblyopia, in which "a high degree of astigmatism was associated with nystagmus and amblyopia and with blue irides."

I now think that my cases and those of Mr. Lloyd Owen were really all instances of incomplete albinism, and that the additional cases given below belong to the same category. But even on this hypothesis the cases are puzzling. Why should all these subjects be males when we know that full albinism may affect either sex? Does full albinism when hereditary, as in the present group, always descend through the mother, even though she be herself unaffected?

Why are all the subjects ametropic, and many of them astigmatic? Pressure by the spasmodically half-closed lids may account for refractive changes in cases where (as in full albinism) there is constant intolerance of light; but there is no such cause in cases of very incomplete albinism.

In what relation does the amblyopia stand to the deficiency of pigment in the eye? Many persons with blonde complexion, blue irides and correspondingly light fundus have perfect sight; even almost complete albinos occasion-

* Lloyd Owen: Ophthalmic Review, i, 239, 1882.

ally have nearly full vision and steady eyes.* But here we have a group of persons who, several years after birth, show a very considerable congenital nystagmus and amblyopia combined with only relative deficiency of colouring matter.

The answer may be that full albinism, even for a short period after birth, prevents the after development of the visual function, even though more or less pigment develops soon afterwards in the retina, and that the cases mentioned above were quite albinotic for the first few months of life: the history however does not bear out this supposition in all of them. Or further, as the pigmentation of normally sighted eyes by no means always corresponds with that of the skin and hair, so it is possible that further search may show variations in the pigmentation of different parts of the same eye—*e.g.*, a translucent iris which impairs the contrast of the retinal images co-existing with a well-pigmented fundus—or even of different parts of the same fundus. Are cases known in which without disease the pigment is wanting at the periphery, though good at the central part of the fundus? and might such a condition explain the occasional occurrence of what seems to be stationary congenital night-blindness without ophthalmoscopic changes? In this connection we may ask whether the light-sense is fully developed in albinos of various degrees? No doubt they are intolerant of strong light, but are their retinæ normally sensitive to very feeble illumination? I do not know whether variations of the light-sense in normal eyes have been systematically connected with differences of pigmentation.

P.S.—Since the above was written, my friend Mr. Herbert Parker, Surgeon to the Central London Ophthalmic Hospital, who has paid more attention than any one else I know to the estimation of light perception, has examined two albinos kindly sent to me by Mr. Lang and Mr. Treacher Collins.

* As in case XVIII, vol. xi, p. 367, of these Reports. Also in the case illustrated in Jaeger's Atlas the patient, a girl of 16, was emmetropic, and read 1 J. from 4″ to 14″.

In Mr. Lang's patient, a completely albinotic boy of 13, the light sense measured by Mr. Parker with his modified Forster's Photometer, was practically normal. There was very high H. As.; V. 6/36 when corrected; quick nystagmus with wide movements.

In Mr. Collins's patient, a male æt. 19, formerly, but not now, fully albinotic (light yellowish hair, no red reflex through the blue irides), Mr. Parker found the light sense reduced to 1/9 in the R. eye and 1/5 in the L., and the light difference perception tested by means of a modified form of Charpentier's Light-difference Photometer, lowered from about 1/10 (the normal) to 8/10 in the R. eye and 4/10 in the L.

This led to enquiry as to night-blindness, and the patient said that he had never been able to see well at night, and was liable to tumble over things, and to pass people whom he would have recognised by daylight.

On March 24, I measured his fields of vision in daylight, using a 5 mm. test object, and Mr. Eason, Mr. Collins's assistant, afterwards measured the right field in the very subdued light obtained by drawing down the almost black blinds—a light just compatible with a full field in Mr. Eason and myself. We found a nearly concentric contraction of both fields, the loss varying from 10° to 20°, and occasionally 30°. The field taken in dull light was decidedly smaller in the nasal half than that taken in full light, the loss being often 25° to 30°. All the observations were carefully made and repeated two or three times for each meridian.

The fundi showed no conspicuous changes, but Mr Collins thought the epithelial surface granular and irregular in texture at the periphery; o. ds. and vessels normal.

V. now 6/18 with each eye with his H. 4 D. corrected; some As., but cylinders do not help. Colour-sense normal. Quick lateral nystagmus.

CASE 1.—*Nystagmus and Imperfect Sight with Blue Irides and H. As.*

Master McC. (9.220), æt. 13 when I first saw him in 1884, had horizontal nystagmus of small range, which was said to have been present from birth; he had always been "near-sighted." His mother had good sight; her father, however, was "very short-sighted," but I do not know what his refractive condition was. An elder brother also was "short-sighted." There was "short-sight" on the father's side too. The patient had slight convulsions between the age of 8 months and 2 years, but this does not seem to bear on the state of his eyes. Irides blue-gray. Hair was never very fair, and is now a light "mousy" brown. Colour-vision normal (Ole Bull's Colour Confusion Table). V. 6/18 and 1 J. at 7″.

He had H. As., and after the use of atropine the condition was:—

R. + 2 D. c. axis nearly vertical
+ 1·5 D. s. } 6/18.

L. + 2 D. c. axis nearly vertical
+ 1 D. s. } 6/12.

Fundi normal, not noted as specially pale, but some superficial (*i.e.*, epithelial) irregularity thought to be visible on nasal side of each O. D. Spectacles ordered for reading. In 1888, æt. 17, I found he had for some time discarded his glasses, because he could do as well without them.

Under atropine now—

R. chose + 4 D. c. axis, as before. 6/12 partly.
L. + 1 D. c. axis, as before. 6/12 partly.

And the spectacles were altered accordingly.

A year later (1889), "the glasses are not of much use." In 1890, under atropine, I found 0·5 D. of M. with the same As, as last time, and V. as before. In 1892 the My. had not increased beyond 0·75 D., and V. and As. were unaltered.

He had proved to have good mathematical ability, and took his degree at Cambridge in the Mechanics Tripos, afterwards passed into the army, and has since been on active service as a signalling officer in one of the Indian frontier campaigns.

CASE 2.—*Albinism of the Eyes with Dark Hair and Well-pigmented Skin. Myopia. Nystagmus. Amblyopia.*

Master C. (26.92), was brought for imperfect sight in October, 1892, when he was 11 years old, and again in July, 1895, when he was 14. The conditions were exactly alike at both dates. He read 4 J. slowly when held very close, chose −9 D. for distance, V. with it being barely 6/60; had slow lateral nystagmus of both eyes, and disliked bright light, so that since he was about 7 he had worn smoked spectacles (−5 D). He was intelligent, and got on pretty well in spite of his imperfect vision. Colour vision quite good (Ole Bull's Confusion Table).

His irides were light gray, and though not actually transmitting the pink fundus reflex in daylight, were translucent; pupils acted well. The fundus in each eye was that of a typical albino, "every choroidal vessel stands out clearly as if there were no other structures, just as in the Plates of the albinotic fundus in Liebreich's and Jaeger's Atlases. In each eye a very large choroidal trunk, (?) artery or vein, passes from the edge of O. D. straight outwards to the y. s. region—indeed in the L. eye this vessel comes from within the border of o. d. as if it had passed up in the trunk of the nerve. The absence of pigment, *i.e.*, the visibility of the choroidal vessels, is perhaps rather *more* marked at the central region than at the periphery."

Notwithstanding this condition of the eyes the scalp hair was dark brown, the eyebrows and eyelashes dark and long, and the skin well pigmented, and his father said that the boy did not "burn" much from exposure to the sun.

The father said that the patient was never at all fair-haired, even as a young child. The eyes oscillated very much at birth; there was never any head nodding. His mother's eyesight was good. Her father (patient's maternal grandfather) had peculiar vision and oscillating eyes; the colour of his hair is not known; his parents were not cousins. No further history can be obtained.

CASE 3.—*Incomplete Albinism with Nystagmus and Imperfect Sight M. As.*

Master Geo. B. (26.152) was brought in November, 1892, at

the age of 4, because he held everything so close to his eyes and saw indifferently at a distance. He had lateral nystagmus, which was at its minimum when he rotated his head to the left and depressed it towards the left shoulder, *i.e.*, when he was looking up and to his right; he adopted this position when looking carefully at objects. His hair was very fair, though far from white; the irides blue or gray and not translucent; the fundi normal, but the choroids very pale coloured from want of pigment; the velvety red of the central region, due to the close meshes of the chorio-capillaris there, well marked. The shadow test, after the use of atropine, showed simple My. As. about 1·5 D. in the vertical meridian, and he was ordered −1·5 D. cylinders.

Two years later, when he was 6, I found his V. to be 6/60 with his glasses, and the same without them if he squeezed his lids together. The shadow test, after the use of atropine, now showed, or seemed to show, considerably more As. and some M., and with correspondingly altered glasses he improved during the next year and a-half, so that when he was between 7 and 8 years old V. was 6/24 and sometimes 6/18. He still invariably turned his head to the left, *i.e.*, looked towards his R., and thus of course looked obliquely through his glasses, this obliquity being increased by another habit he had acquired of pulling the right side of the spectacles forwards. The refraction again measured, under atropine, in February, 1897 (æt. $8\frac{1}{2}$), showed My. As. 3·5 D. in L. and 4 D. in R., and I ordered −4 D. cylinders, though he saw no better with them than with the somewhat weaker ones he had been using. The R. eye, separately tested, now seemed defective (6/60 ?); with L., and with both together, V. was 6/24. Subsequently +4 D. cylinder was ordered, in addition, for reading, as he preferred it. In this way he got on fairly well, and when I last saw him, in May, 1901 (æt. 13), V. was 6/18 partly with −4 D. cylinders, and he was doing well at school. His hair was now a dull, flaxen colour ("tow colour"). He was well-grown, strong, and keen, though of course backward in books. Another oculist has examined him again since then, and finds the refraction not very different from my first result in 1892, and has accordingly reduced the cylinders, V. being still 6/24; he also finds that V. is equally good in R. and L.

This boy's eyes were noticed to "work" a few days after birth; there was no "head nodding," he had no fits, and his health as an infant was good. His hair was white at birth, and his skin was, and is, extremely fair, but he has become liable to freckles. His eyes (pupils) were never "pink." He had influenza badly when 3, and again when 7 years old, with resulting cardiac weakness, and all attempts at book learning were forbidden by his physicians till he was about 10, but the heart is reported now to have recovered.

He is the fourth child; was born at full time, and his mother was quite healthy when carrying him. No. 1, a boy, 8 years older than the patient, is said to have been born with dark hair, which turned fair, and to have blue eyes and fair skin; No. 2 (girl), also born with dark hair, has blue eyes and dark skin; No. 3 was premature, and died in infancy.

The amount of ametropia, and especially of the astigmatism, diagnosed in this boy's eyes varied a good deal at different examinations, but I think these results were due chiefly if not entirely to the patient's habit of rotating and tilting his head in such a way that he always looked upwards and to his right. The effect of the resulting obliquity of his line of sight in relation to the plane of his (concave) lenses would be to increase the power of the lenses required to turn the shadow (unless these could be held in a correspondingly oblique position), and to diminish in a similar manner the strength of spectacles placed in the usual position on the face. The exact amount of this alteration could not be calculated accurately in his case,* because his spectacles were sphero-cylinders; his line of sight did not correspond to either of the chief meridians of the lenses, and the exact degree of obliquity that he employed no doubt varied in different circumstances.

CASE 4.—*Incomplete Albinism. Oscillation of Eyes and Head. Subsequent Pigmentation of Hair, but no Improvement of Eyes. Hypermetropia.*

Master B. (26.232) was sent by Dr. Russell Dodd in January, 1893, when 11 years old. His hair was fair, but far from white, pupils slightly pink, irides light blue and translucent on ophthal-

* See Ward Holden on "The Cylindrical Equivalent of Tilted Lenses," &c. Tables III to VI, Knapp's Archives, xx, p. 2 1891.

moscopic examination. At the equator and periphery of the fundus in each eye every choroidal vessel was seen with absolute distinctness, owing to complete absence of pigment, and apparently (but of course only apparently) of chorio-capillaris; over the central area, well towards, though not reaching quite up to, the equator, the fundus was of the normal velvety bright red, no choroidal trunks being visible. This full-red area was much larger, and the transition from it to the peripheral zone of visible trunks much more abrupt than is shown in Jaeger's Atlas, my note being that—"The difference between central and peripheral regions as regards apparent capillary supply of choroid is extreme."

The patient was backward in reading, and needed large print. I found he could read up to 8 J. by holding it very near, but nothing smaller, and V. was less than 6/60. Refraction considerably H. There was rather quick lateral nystagmus of moderate range, and when reading, his head also oscillated laterally; I thought this was not so quick as the oscillation of the eyes, but could not be certain.

After the use of atropine the shadow test showed in

R. H. 4·5 with 0·5 to 1 As.
L. H. 6 with ? 0·5 As.

and he was ordered + 3·5 D. for reading.

In March, 1899, æt. 17, he was "getting on pretty well," was strong and well grown, but had a nervous, agitated manner. The nystagmus, oscillation of head, and condition of the eyes were in every particular exactly as before, neither the irides nor the fundi being any more pigmented, but his hair was now decidedly brown.

V. R. Hm. 3 d. with { + 3 s. / + 1 c. } 6/36, well.

L. H. 4 d. { with + 4 D. s. / with or without + 1 c. } 6/36, not so well.

In this case the nystagmus had not been noticed till the age of 6 months, when the boy had whooping cough. His hair was almost white as an infant, but gradually became coloured. He was the second of four children. No. 1 died of whooping cough; Nos. 3 and 4 have good eyes and nearly black hair.

IV. Case of Uniocular Spasm of Accommodation.

There seems to be no weak point in the evidence in the following unusual case.

Miss O., æt. 21 (48.134), came on November 21, 1898, complaining of defective sight in her R. eye. She had found it out about a week previously, when she had resumed the use of her typewriter after a long rest. She was quite aware of the defect when both eyes were open, and it annoyed her.

I found the visual acuity and refraction as follows:—

R. 6/60, chooses very definitely − 3 D. 6/5; unaided reads 1 J., best at 6″. Fundus normal; refraction estimated H. 1 D. at O. D. but Acc. constantly varying; the eye sometimes has little fits of fine tremor when under ophthalmoscopic examination. Shadow test shows H. L. 6/9 slowly, Hm. at first 1 D., on second trial 2 D., 6 5. Unaided reads 1 J. best at about 12″. Ophth. n. Refraction estimated H 2 at O. D. Pupils not noted.

For reading 1 J. comfortably and without sense of difference between the two eyes she requires

$$\text{R. } -1{\cdot}5 \text{ D.; L. } +1 \text{ D.}$$

After atropine for two days:—

$$\text{R. } 6/18: \left\{ \begin{matrix} +1 & \text{D. s.} \\ +0{\cdot}5 & \text{D. c.} \end{matrix} \right\} 6/5; \quad \text{L. } 6/18 \left\{ \begin{matrix} +1 & \text{D. s.} \\ +0{\cdot}5 & \text{D. c.} \end{matrix} \right\} 6/5.$$

Ordered full correction, for all near work; no glasses for distance.

A year later (November 13, 1899) Miss O. came again for pain in R. eye of a few days' duration; V. at first was R. 6/24, but after some waiting it improved to 6/9:

$$\text{and with } \left\{ \begin{matrix} -1 & \text{D s.} \\ +0{\cdot}5 & \text{c.} \end{matrix} \right\} 6/5 \text{ slowly.}$$

L. 6/5 easily at once; better with + 0·5 c.

Pupils equal and acting fairly.

R. palpebral fissure was rather narrower than L. as if from slight spasm, but no tremor was visible.

This young lady was highly skilled and very quick in shorthand and typewriting; she worked intensely at whatever she did but had not much staying power; her mother told me the patient had always been subject to twitching movements of the face, but none were noticed when she was with me.

IV. Case of Slight Sore on Finger causing Axillary Abscess. Then, one-sided Anterior Headache and Rapid Failure of Corresponding Eye, and soon after of other Eye, from Neuritis leading to Atrophy. ? Septic Inflammation in front of Chiasma.

Mrs. W., æt. 42 (52.135), was sent by Dr. Florence Orford, of Pontefract, in February, 1900, nearly blind from atrophy of the optic nerves. I saw her on 13th and again on 22nd.

The R. had no sight except in the upper-inner part of F. where she could see my hand. P. showed very little direct reflex action. O. d. pale, yellowish, very slightly hazy, vessels rather smaller than in L., a few slight dots about y. s., but no signs of previous œdema either of o. d. or retina.

With L. she saw 6/36 and sometimes 6/24, but found the letters slowly owing to the very small size of F., of which only a sector passing from the centre upwards and inwards remained, exactly symmetrical with the surviving remnant in R. P. always smaller than R. and acting fairly well to light. O. d. less pale than R., veins rather large, arteries normal. Some white lines along the veins on both discs.

No oculo-motor paralysis. Hearing good, but the sense of smell (iodoform) not at all prompt.

She was a pale rather sallow woman, had been married 12 years, had one child, æt. 11, and had had no other pregnancies. She and her husband had a small house and let lodgings. The history, obtained from her own account and from notes supplied by Dr. Florence and Dr. John Orford and Mr. Adolf Bronner, was as follows :—

She had been in poor health and had suppression of menstruation all the summer of 1899, though suffering from nothing that took her to her doctor, when, about the end of September, she had a small "festered sore" on the dorsum of R. index finger, which, however, did not prevent her from attending to her house work as usual. It soon healed but was followed by a swelling in the axilla, for which she saw Dr. Florence Orford, on October 26. This rather slowly matured into an abscess with

gradual rise of temperature, during part of which time she was ill enough to be in bed. Dr. John Orford opened the abscess (about one ounce of pus), and temperature gradually became normal. Several weeks passed between the commencement of the glandular swelling and its maturation.

Either shortly before or soon after the abscess was opened, she began to have headache, and within a few days of the operation she complained of severe pain on turning the L. eye out, and had to turn her head when she wanted to look to her L. The pain then extended to, or became localised in, the L. frontal and temporal region, and, whilst she was still in bed, the sight of L. eye failed quickly. The eye became absolutely blind for a few days and then recovered some sight. The patient said that the L. again failed and again recovered, but this is not confirmed by the medical notes. However this may have been, about 10 days after the first failure of L. the R. eye failed rapidly in a similar manner, and as the patient was then convalescent she was sent, on December 18, to Dr. Adolf Bronner, at Bradford. This was 10 to 12 weeks from the occurrence of the sore on the finger, about 5 or 6 weeks from the incision of the abscess, say 3 weeks after failure of the L. eye and exactly 6 days after failure of the R.

When admitted at the Bradford Eye Hospital, the conditions found by Dr. Bronner were :—

R. no p. l. ; O. D. nearly normal.
L. 6/36 ; O. D. showing signs of subsiding papillitis.

She was put on mercurial inunction and iodide of potassium and kept in for a month, during which time both o. ds. became pale.

Headache seems to have ceased whilst in hospital and did not recur. The patient says she never lost her smell and had no discharge from the nose. She believes she was deaf for a short time whilst in the hospital, but Dr. Bronner did not mention this in his note to me. The urine examined at the commencement of the axillary abscess was free from albumen and sugar. There were not at any time the slightest indications of constitutional syphilis ; nor was the course of the sore on the finger like that usually taken by a chancre. No reason to suspect glanders. There were neither signs nor symptoms of orbital

inflammation, or affection of oculo-motor nerves; for the pain in turning the L. eye outwards and compensatory rotation of the head might be explained by localised inflammation of the sheath of the optic nerve without paralysis of the external rectus.

There seems no reason to doubt the dependence of the optic neuritis and blindness in this case upon the illness due to the axillary abscess; but whether the optic nerves were poisoned by a toxic condition of the blood, or affected at their junction with the chiasma by inflammation of the underlying periosteum or bone, cannot, I suppose, be settled. On the latter hypothesis septic embolism may be supposed to have occurred on a small scale. The surviving remnants of the visual fields—of the temporal hemianopic type—would be compatible with disease at the anterior central part of the chiasma or the adjoining parts of the optic nerves at their emergence. Nothing in the case suggests thrombosis of venous sinuses, nor was there any positive evidence of suppuration in the sphenoidal sinus.

P.S.—January, 1902.—I hear from Dr. Florence Orford that Mrs. W. is now in good health, and her sight considerably better than it was two years ago. The outer part of R. f. is wanting, but she can read ordinary print with each eye, with her +1·5 D spectacles and can see to do her housework.

V. Case of severe Single Retrobulbar Optic Neuritis leading to Complete Atrophy. Commencing Sarcoma of Suprachoroidea near the Yellow Spot in the corresponding Eye.

Mrs. ——, 40 (45.180), was sent by Dr. Pigeon, of Hull, on February 22, 1898. Incipient tumour of the choroid had just been diagnosed in the R. eye by an ophthalmic friend in London, and a second opinion was sought.

It may be mentioned at once that ophthalmoscopic changes resembling the earliest stage of choroidal growth near the y. s., co-existed with symptoms and appearances pointing to retrobulbar optic neuritis or some other acute process in the nerve. The co-existence of such apparently unrelated conditions much increased the difficulty of deciding what course to recommend.

Mrs. —— gave the following history:—On Sunday, February 6 (16 days before I saw her), she had pain in the R. forehead, top of the orbit, and behind the eye, with watering of the eye and some swelling of the lids, especially the upper lid; the pain was present chiefly during movements of the eyeball. She was seen by Dr. Pigeon on 10th and 14th, and by Mr. A. L. Roe in consultation on 17th. She began to notice that the sight of this eye was failing between 10th and 14th; at first she could see the doctor's forehead when she could no longer see the lower part of his face, this and other tests all showing that the lower part of the F. failed first; by the evening of 17th the eye had become totally blind, and it had remained so ever since.

When I saw her on February 22 there was still some pain in movements of the R. eye; no displacement of eye, no swelling of lids, and no restriction of movements; T. n.; there was the barest p. l.; pupil dilated from atropine; media clear; o. d. perhaps slightly pale but not hazy as compared with L.; veins rather beaded on o. d., and arteries rather smaller than in L.; slight spontaneous pulsation both of arteries and veins on the o.d. A large circular patch of the choroid above the macular region was of a yellowish-gray colour, somewhat mottled but not showing any visible choroidal vessels, slightly raised (H. 3 D. at the

middle or most prominent part, against Em. at y. s.), the lower border well defined, the upper border passing more gradually into healthy choroid. The area of this patch was from four to five times that of the o. d. It was crossed near its lower part by the upper temporal vein, and higher up by the corresponding artery; it was quite above the upper macula artery. The overlying retina was silvery, reflecting too much light, and showed slight undulations of surface but no proof of detachment. No hæmorrhages anywhere. Other parts of fundus n. The other (L.) eye perfect in every way and Em.

The patient, a rather florid lady, had a baby 4 months old, which had been suckled for the first 6 weeks only; she had menstruated once since the confinement. She had had influenza slightly some weeks after the confinement. She had been married 10 years; six pregnancies, the first two ending in miscarriage, the last four living and healthy; there was nothing pointing to syphilis. The family history pointed to tubercle:—a maternal aunt died of "consumption," æt. 16; Dr. Pigeon had lately operated on the daughter of the patient's only sister for tubercular glands; and the patient herself when a girl had been thought to have one lung weak, and was much taken care of for several winters. The patient's father died of drink; he had only two children, the patient and her elder sister.

With the above history and the very unusual relation between the onset of the eye-symptoms and the ophthalmoscopic changes, I thought it right to wait and treat for a time by mercury; but I sent her to Mr. Marcus Gunn before deciding. He also advised waiting for a time and trying the effect of treatment, including cod-liver oil. She was accordingly put on mercury, and I believe took cod-liver oil regularly. Dr. Pigeon wrote that on March 16 (4 to 5 weeks after the sight failed) the patient had noticed slight return of vision in the lower part of the F., and that this extended over the upper part next day. On 18th he found she could see movements of the hand, and on the same date he observed slight tenderness of gums. Dr. Roe examined the fundus and found the patch certainly no larger.

On *April* 1 I saw her again, mercury having been continued with little or no intermission, but so as to avoid salivation. There was then no central vision, but she could count fingers

held about 30° below the centre of F.; and could see the hand at the periphery, except over a considerable area in the upper-outer part, which was blind. The blind part just below the centre, scarcely agreed with the situation of the choroidal patch; and the loss of the upper-outer part of F. had not any, even approximate, counterpart at the fundus; p. acted to light; t. n. The choroidal patch was no thicker, indeed I thought it rather less H.; it was less uniform in appearance, more mottled, and the retina now showed little or no watered-silk reflection; the o. d. had become uniformly pale all over and was clear.

May 20.—R. cannot now see fingers anywhere, but the amount of absolute loss of F. does not seem larger; choroidal patch certainly more mottled, and perhaps more pigmented, but H. now to me only 1 to 1·5 D where formerly it was 3 D. O. D. now very white and perfectly clear, and one of the retinal arteries considerably constricted.

July 28.—Now no p. l. in any part of F.; thinks all sight went about a month ago. No fresh symptoms or appearances, but I estimate the highest part of the patch at H. 3 again, as at the first examination.

On August 2 the eye was excised, the optic nerve being cut at least 12 mm. behind the globe. Sections were made by Mr. Devereux Marshall, and I believe they were in the plane of the vertical meridian. Microscopical examination showed the choroidal thickening to have the structure of a sarcoma. The growth had evidently begun in the lamina fusca, with which its outer aspect was closely incorporated, and by which it was adherent to the sclerotic; indeed the sclerotic was somewhat thinned over the deepest part of the growth, though no trace of infiltration of its fibres could be found. The choroid itself was natural (except for turgescence of its vessels) at the margins (thinner parts) of the growth, but over the central (thicker) parts it was much atrophied and more or less infiltrated by the tumour cells; the elastic lamina was, however, intact everywhere, and the retinal pigment epithelium, though thinned and its nuclei diminished in number over the centre of the tumour, could be followed without a break quite across. The tumour cells were oat-shaped and in cross-section had almost exactly the diameter of a red blood-corpuscle; in the outer (scleral) part

they stained deeply with hæmatoxylin, and were very closely packed; the inner layers were less stained and more loosely packed. There was more pigment in the growth, especially in its inner layers, than the ophthalmoscopic appearances had led me to expect; it lay partly within the cells, but was partly the normal pigment of the suprachoroidea scattered amongst them.

The retina generally showed atrophy of its nerve-fibre layer; the other layers appeared to be healthy, except on a patch measuring from one-third to one-half the diameter of the tumour, and corresponding to its centre, *i.e.*, thickest part; here the bacillary layer and the outer granules had disappeared, and the outer molecular layer was represented by an open meshwork of fine fibres (Müllerian) containing scattered round cells, some of which were pigmented; this change in the retina over the centre of the growth was found in all the sections that included the centre. The tumour measured almost exactly 1 mm. in thickness at its deepest part, and thinned away quite gradually on both sides into healthy choroid. Its border was nowhere nearer than about $2\frac{1}{2}$ disc breadths to the margin of the o. d.

The optic nerve was atrophied: no healthy nerve fibres could be made out; the connective tissue trabeculæ were much nucleated and thickened, the small blood-vessels enlarged, and the inner (neural) layer of the arachnoid sheath irregularly thickened by deposit of cells, with oval nuclei larger than and quite different in appearance from the cells of the tumour.

It seems on consideration impossible to connect the tumour with the destructive retrobulbar neuritis in this case, and we must assume that these two rather infrequent conditions occurred in the same eye independently of each other, and that the tumour would not have been discovered till much later, but for the loss of sight caused by the neuritis. The small patch of degenerated retina over the centre of the growth must presumably have caused a corresponding defect in the field; but this retinal change was doubtless a gradual matter, and even had it occurred rather rapidly, owing to a corresponding advance in the growth, it would not explain the very extensive loss of field that took

place within 3 or 4 days. That this primary loss of field corresponded roughly with the situation of the new growth can have been only a coincidence. Even if the acute inflammation in the optic nerve set up collateral hyperæmia of the tumour, and thus led indirectly to some degree of œdema of the retina lying upon it, such as the early ophthalmoscopic appearances indicated, we cannot assume that the function of the swollen portion of the retina would be abolished unless the œdema led to microscopical degeneration co-extensive with the early loss of field; and such was not the case, for, as already stated, by far the larger part of the retina lying on the tumour was (with the exception of the atrophied nerve-fibre layer) microscopically sound. It is perhaps not impossible that a rapidly forming secondary growth in the bone or periosteum at the optic canal might have set up the optic neuritis, but gradual atrophy from the pressure would have been the more likely result of such a complication; and, moreover, there was not the slightest ground for supposing that secondary growth had occurred either in the orbit or anywhere else.

P.S.—January, 1902.—Dr. Pigeon informs me that the patient is now (three years and a half after removal of the eye) perfectly well, and the orbit quite healthy.

CASES OF MESOBLASTIC TUMOURS OF THE ORBIT.

By A. Quarry Silcock and C. Devereux Marshall.

Case 1.—William Arnold, æt. 28 (4198), a stonemason was admitted to the R.L.O.H. on January 11, 1894, with the following history :—

History.—About 3 years ago the patient noticed a swelling about the size of a pea at the outer part of his orbit. He was sent in for immediate operation.

A tumour was felt about the size of a small chestnut in the upper and outer quadrant of the left orbit, causing a considerable swelling beneath the skin. It was freely movable and tense. Its anterior surface was more rounded than its posterior.

A horizontal incision was made over the tumour, and by dissecting down its surface was exposed; it had a fairly well-defined capsule, but it was attached behind by a pedicle; this was cut and the wound closed.

Path. Report.—Encapsuled growth of firm consistency, 20 × 17 mm.

Microscopic Examination.—The growth is composed of irregularly-arranged fibrous tissue. At one part there is a series of sections of gland acini, some of which are dilated; others are simply lined with columnar epithelium, and resemble normal lacrimal gland. The growth is a fibroadenoma.

Case 2.—Samuel Herbert, æt. 32 (70, O.P.), was admitted on September 16, 1895, with a history that the left eye commenced to fail 3 or 4 months previously, and more recently the right eye has become defective. There is no history of injury or of syphilis.

Present State.—The left eye is pushed down and out: the media are clear, there is well-marked optic neuritis, the vision being 4/60 J. 18: movements upwards defective. Right eye, slight optic neuritis, V = 6/12 J i. Ordered, Pot. Iod., gr. x, and Ung. Hydrarg.

He was readmitted on December 12, 1895, for although the

treatment had been continued, yet there was no improvement in the condition.

On readmission the left eye was found to be much proptosed and displaced downwards and outwards, and the upward movement was interfered with.

There was a distinct swelling beneath the outer portion of the root of the orbit, which felt tense and elastic. The optic neuritis was well marked in this eye, and the disc in the right eye was very similar to what has previously been described. The left V. = 6 24 J. 8.

On December 12, 1895, the growth was cut down upon. It consisted of soft granular material, some of which was scooped out. There was considerable hæmorrhage, which was controlled with plugs of cotton wool soaked in turpentine.

He was not again seen until 1898, when he was readmitted on May 5. Two weeks previously the eye became suddenly painful and watery without any injury or other circumstance to account for its doing so.

Present State.—There is very marked proptosis and injection of the left eyeball, which is pushed down and out, the movements being greatly limited. A mass can be felt in the orbit. There is a large ulcer on the cornea; the iris is discoloured and the pupil inactive. V. = P.L. only.

On the following day the contents of the orbit were removed. This healed well and patient was discharged on May 25.

He has been seen at intervals since, the last time being February 1, 1901, when there was no sign of recurrence. The vision of the R. eye was 6 6 partly. The field was full, but the disc retained the same blurred appearance suggestive of neuritis which was first noticed in 1895.

Microscopic Appearance of Growth.—The growth is composed almost entirely of cells. Some parts of it are solid, the cells being packed together with little or no reticulum. At other places there are an enormous number of spaces, some of which are empty, but others contain hyaline material. Scattered through the growth are numerous vascular spaces, which contain blood, but there are no well-formed vessel walls. The cells are for the most part large, and contain single large nuclei, with but little protoplasm.

Case 3.—Mary Carr, æt. 53 (40., O.P., 4652), was admitted on July 3, 1895, with the following history :—

On February 25, 1886, patient was admitted under Mr. Streatfield suffering from "tumour of the orbit," which had existed for 12 months. "The right globe and contents of the orbit were cleared out; a soft lobulated growth removed with globe." Nothing of the nature of a solid growth was discovered by the pathologist.

She complains now of having suffered severe pains in R. side of forehead and vertex for more than a year, but has noticed a swelling in the orbit for only 6 months. This has been slowly getting larger and the pain more severe.

Present State.—There is a large rounded swelling about the size of a walnut occupying the right orbit. It is tense and has distinct pulsation, but no bruit can be heard over it. The mass reaches the outer wall of the orbit, but this portion is not prominent. The skin over the tumour is freely movable, and covered with dilated veins.

The following day an incision was made over the growth and the skin reflected; an attempt was then made to shell out the prominent part of the growth, but this was found to be impossible, as it extended backwards probably into the cranium. There was much bleeding, which was arrested by packing with gauze soaked in turpentine. A portion of the growth was removed for microscopic examination.

The temperature quickly rose, and patient became very ill. There was no optic neuritis in the left eye. A mitral murmur developed and she became comatose, and died on July 18.

An autopsy was made (42, O. P.).

The growth is composed of cells having rather large round nuclei. Mixed up with it is a considerable amount of pigment, the greater part of which is altered blood. Numerous areæ of hyaline degeneration are present, and the tumour is extremely vascular. It is a myxo-sarcoma.

Case 4.—Benjamin Armitage, æt. 41 (78, O. P., 4879), was admitted on January 23, 1896, with the following history :—

In October, 1895, he was first seen as an O. P. suffering from proptosis of the right eye with diplopia; this had been noticed about a month.

The only history of injury was about 5 years previously, when he was struck beneath one or other eye by a splinter of wood which penetrated the skin. There is no history of acquired syphilis, and no reason to suspect congenital syphilis.

Present State.—There is considerable proptosis of the R. eye with upward deviation of the globe; the downward movement of the eyeball is much interfered with.

Within the lower margin of the orbit a firm rounded swelling can be felt. Some dilated lymphatics are seen on the lower lid, as well as tortuous vessels on the sclera. The inner margin of the disc is slightly blurred, but the vision in each eye is 6/6 J. i.

He was first treated with mercury inunction and iodide of potassium internally, but without effect.

On February 7 a portion of the growth was excised, and it appeared to be connected with Tenon's capsule.

The mercury and iodide were continued, and patient was discharged on April 16. He was again admitted as the growth was evidently increasing in spite of treatment.

There was then found to be slight optic neuritis, and the vision with correction was only 6/18 J. 6 with the right eye, though it was normal in the left.

On April 17 the whole of the contents of the orbit were removed, which included a large growth which reached to the apex. The bone was scraped, and the apex, where apparently some growth remained, was burnt with the cautery. The patient did well.

He was not seen again, but information was received to the effect that he died on January 5, 1898, after a long and painful illness, during which the tumour spread to the other eye and to the spinal cord, causing blindness, paralysis, and great suffering.

Microscopical Appearance of Growth.—The growth consists of numerous small round cells and fibrous tissue, together with muscular fibres which are surrounded by the growth.

There are some bands of fibrous tissue which intersect the growth and partially surround it, as well as numerous spaces which contain blood and probably represent badly formed vessels.

The growth is a small round cell sarcoma.

Case 5.—William Graham, æt. 48 (4967), was first seen in April, 1894, with a "lipoma-like swelling" beneath the left upper lid, which had been noticed about a month. When 15 years of age, he had an iron-headed arrow shot into the left eyebrow, but this was removed entire.

On May 29, 1894, the growth was found to be adherent to the lacrimal gland, and the greater part of it was removed.

In February, 1895, the growth had recurred, and it was again removed, and found to extend deeply into the orbit.

On July 23, 1896, he was readmitted as the growth had increased much in size during the last few weeks.

Present State.—There are no enlarged glands to be felt. The growth is much larger than it was before, and is a semi-solid glandular-like mass which overlaps the cornea above, leaving only the lower portion uncovered. The globe is proptosed and displaced downwards, and its movements are almost completely abolished. The growth is adherent to the globe and the lower portion of the cornea is abraded.

On July 24, the whole contents of the orbit were extirpated. A large mass of growth was found to be filling the roof of the orbit, and extending backwards to the apex to which it was firmly adherent. Elsewhere it was removed clean with the periosteum.

Patient has remained in good health ever since, and when last seen, on November 28, 1900, there was no sign of recurrence, and he was in good health.

Microscopical Examination.—The growth consists to a very large extent of fibrous tissue; there are also small round and spindle-shaped cells. Numerous muscle fibres are seen in the section which are separated by the fibrocellular tissue of the growth. The orbital fat is largely infiltrated, and the blood vessels are large and well formed.

The growth is a fibro-sarcoma.

Case 6.—George Draper, æt. 60 (5016), was first admitted on February 28, 1888, with the history that 35 years ago a growth commenced in the left eye which was removed at Guy's Hospital, where he was told it was "fatty." The present lump has been noticed for 1½ years, with a rapid increase during

the last 2 or 3 months. An incision was made and the mass removed.

He was again seen on November 21, 1893, with the history that since the last operation things remained quiet for 2 years and then recommenced growing. Again it has been removed.

On September 21, 1896, he was admitted with a large swelling in the region of the left inner canthus. It was lobulated, and separated the lower lid from the globe. In places the mass was hard and adherent to the bone, but at other places it was soft and cystic. The globe was displaced upwards; but the eye retained good movement.

An incision was made over the tumour and a portion removed. The growth was extensive and invaded the spheno-maxillary fossa and the superior maxillary bone. It was removed as far as possible; but the eye was not touched.

Patient was not again seen nor heard of.

Microscopic Examination.—The growth consists of small round and spindle-shaped cells, containing a stroma which can be traced between each individual cell.

There are numerous spaces which are evidently blood vessels, but they have no well-formed vessel wall.

The growth is a mixed, round, and spindle-cell sarcoma.

Case 7.—Charles Love (5614), aged 9, was admitted on December 12, 1898, with the following history. Six weeks previously he had a blow in the right eye with a football. The next day a swelling was noticed which has increased in size since.

Present State.—Between the globe and the inner extremity of the R. lower lid is an irregular round and painless swelling extending from the margin of the orbit backwards. It is about the size of an almond. On everting the lid, the conjunctiva has a dark appearance in the cul-de-sac.

R. V. = 6/24 partly Cyl. − 2·0 70° 6/18.
L. V. = 6/24 partly Cyl. + 2·0 90° 6/12.

On December 13, 1898, the growth was removed, and the wound healed. The patient was last seen on February 1, 1901, when there was no sign whatever of recurrence R. and L. V. − 6/12.

Microscopic Examination.—The growth consists of numerous small round cells which are crowded together and contain but little stroma. No well formed blood vessels are visible, and there are but few strands of fibrous tissue running through the growth. It has the appearance of a rapidly-growing round cell sarcoma.

Case 8.—Alice Bayley, æt. 28, was admitted on July 12, 1897, with the following history :—

The left eye had become prominent for 16 months past. There was no pain, no history of injury, and no nasal trouble.

Present State.—The left eyeball is displaced forwards and somewhat downwards. The movements of the eyeball are free in all directions. There is a hard swelling at the inner and upper part of the orbit. L.v. = 6/6.

On July 13, the mass was cut down upon, and proved to be a new growth, a portion of which was removed, when microscopic examination showed it to be an endothelioma.

She was again admitted on October 3, 1898. The swelling was found to be much the same, and there was considerable frontal headache, and the L.v. = 6/18 partly.

There was marked proptosis ; the movements of the eyeball were free.

At the inner extremity of the roof of the orbit was a hard irregular swelling, which protruded slightly, and extended backwards into the orbit. No glandular enlargement.

No operation was performed.

She was again admitted on January 16, 1899. At this time there was marked proptosis of the left eye, and slight displacement down and out. Convergence was slightly interfered with. Extending inwards from about the middle of the roof of the orbit is an irregular hard mass, which yields on pressure and causes pain ; it extends backwards into the orbit. The left optic disc is pale and well-defined. L.v. = 6/36.

On January 17, an exploratory incision was made, and a probe could be passed backwards through the growth.

On January 20, the contents of the orbit were removed, and the patient did well.

On June 1, she was again admitted, as there was some

evidence of recurrence in the socket. This was readily removed.

She was heard of in April, 1899, at which time the doctor reported that the growth appeared to be recurring.

She has also been heard of in December, 1901, and the report states that but for a little discharge from the socket pit, is well and in fairly good health.

Microscopic Examination.—The growth consists of cells which are more or less round and spindle shaped, together with numerous clumps of what appear to be degenerated and compressed cells, somewhat resembling the cell nests in an epithelioma.

At places some pigment is seen, which forms part of the growth.

? Endothelioma.

CASE 9.—Mr. S., æt. 35, was first seen suffering from a proptosis with some swelling and a hard mass to be felt at the outer and lower part of the right orbit.

An exploratory incision was made, and a soft solid mass was entered, which was very vascular. A portion of this was removed, and after hardening, was submitted to microscopic examination, when it was shown to be an endothelioma.

On February 28, 1900, the eye, together with the whole contents of the orbit, were removed, leaving only the lids.

A large mass was then found at the outer and lower part of the orbit, not involving the lacrimal gland, but extending far back into the apex. Every piece of the soft tissue of the orbit, together with the periosteum, was removed.

The patient made an uninterrupted recovery, and now, in February, 1902, he is in perfect health, and there is no sign whatever of recurrence. The further portion of the growth was examined by Mr. G. H. Plimmer and Dr. Sidney Martin, who agreed with the diagnosis.

The cases here recorded of primary tumours of the orbit have been brought together partly with a view of determining more exactly the prognosis in this important class of case, and partly because several of them present features of interest.

In vol. xiii of the Ophthalmic Hospital Reports, Stirling recorded 29 cases of sarcoma of the orbit, and of these the prognosis was extremely bad, and left only a percentage of about 17 who had passed the three-year limit, although the cases whose history had been followed up showed that recurrence and death may take place at a much longer interval.

Sex.—Of the 9 cases here recorded, 7 were men and 2 women. This is a much higher percentage of men than Stirling found, for out of 29 cases, 14 were males and 15 females.

Age.—Of these cases the oldest was Case 6, a man æt. 60, while the youngest was Case 7, where the patient, a boy, was 9 years old. The average age of the patients was 38 1/9 years, while in Stirling's cases it was 28 2/12 years.

Side.—The growth occurred in the left orbit five times and in the right four times.

The known duration of the symptoms previous to operation ranged from six weeks in Case 7, to 10 years in Case 3, while in one case, No. 6, a tumour of the orbit had been removed 45 years before the last operation was undertaken, the result of which is unknown, the patient not having been seen or heard of since leaving the hospital.

Result.—Of the nine cases, five are known to be alive, two are dead, and two are unknown.

Recurrence.—Case 1 unknown.

Case 2. No recurrence after removal of contents of orbit in 1898.

Case 3. Patient died 15 days after operation. The growth was not removed, as it was found to extend into the cranium.

Case 4. Patient died 19 months after removal of contents of orbit of general recurrence.

Case 5. Recurred a year after partial removal, and again a year later after further removal: no recurrence since.

Case 6 was seen first in 1888. The contents of the orbit were partially removed in 1852. The recurrent mass was

excised in 1888. It reappeared in 1891, and in 1893 it was again removed.

In 1896 a large mass was present, which, on exploring, was found to involve the spheno-maxillary fissure and the superior maxilla. It was impossible to remove it, and the patient was never seen again.

Case 7 has not recurred since the growth was removed.

Case 8. The growth was partially removed in July, 1898, and the contents of the orbit were removed in January, 1899, since which time there has been no recurrence.

Case 9. Orbit cleared on February 28, 1900, since which no recurrence has taken place.

The operation of extirpation of the orbital contents, if done subperiostally, is simple, and we prefer to do it in the following manner:—

The edges of the lids, including the Meibomian glands, the hair follicles, and the whole of the conjunctival sac, are separated from the surrounding parts down to the bone. A periosteal elevator is then run round the edges of the orbit, and with this instrument the entire orbital contents are separated from the bone as far back as possible. The optic nerve, together with a small amount of surrounding tissue, is divided as near the optic foramen as possible, and the whole of the orbital contents, including the eye, are removed *en masse*.

As the parts cicatrise, the remains of the lids and the surrounding skin are drawn into the orbit, which becomes completely lined by skin. If the lid margins and conjunctival sac are left, these structures are not only useless, but they leave a mucous discharging surface or cavity at the back of the orbit which is a source of discomfort to the patient.

We strongly disapprove of the use of caustics, such as chloride of zinc paste, which are sometimes placed in the orbit after partial removal of its contents. We consider the practice to be both painful and dangerous, and certain it is that what cannot be with safety removed with the knife and chisel, possibly aided by the Pacquelin's cautery, had better

be left behind; clearly it is far better to know the full extent of the operation which is undertaken than to trust to a powerful chemical caustic which destroys an unknown amount of tissue.

The deformity after total extirpation of the orbital contents is, of course, great, and cannot be remedied; probably the best thing to do is simply to cover up the cavity with a celluloid shield attached to a spectacle frame.

That these tumours differ very much in character is at once obvious, and in the present state of our knowledge the origin of many is obscure.

Amongst the most interesting are those described as endotheliomata. Much has been written on this subject by Van Duyse, Coppez and others, tending to show that they have certain characteristics which lead one to suppose that they are derived from the endothelium lining the lymphatics of the orbit.

All that have been observed by us are when cut into often very soft and vascular. As a rule, as soon as the tumour has been incised there is considerable hæmorrhage, but with a sharp spoon some of the growth can be safely scooped out and prepared for histological examination.

No matter how free the hæmorrhage may be at the time there is no difficulty in stopping it with pressure.

They are unencapsuled, and the growth infiltrates the normal tissue of the orbit, and may be seen pushing its way through and separating such structures as muscle fibres, &c.

Endotheliomata show very marked local malignancy; that is to say, they have continued to grow so long as any of the tissue is left behind, but when once this has been thoroughly cleared out they have done well. So far as we have seen there has not been any marked tendency to generalisation, and when once the growth has been completely removed the patients have remained in good health, giving no sign that they were suffering from any visceral complication.

Very different are the result in cases of sarcoma, whether

pigmented or white, which occur both in the orbit and in the eyeball. For these, as was shown by one of us in the last part of the Ophthalmic Hospital Reports, the number of patients who died of general recurrence was large, and amounted to 58·62 of the cases which were traced.

Time, however, will prove this point, provided all the cases are strictly examined.

In conclusion we would urge the importance of recording such cases, for without a large number from which to draw conclusions, errors in diagnosis and prognosis cannot be eliminated.

ARCUS SENILIS.

By J. Herbert Parsons, Curator.

Very various opinions have been expressed from time to time as to the pathological changes in the cornea in arcus senilis, as will be seen from the following historical *résumé*:—

Duddell (1): "I take this opacity to proceed from the dryness of the parts: the pores being too close do not admit a sufficient quantity of fluid into the parts to maintain their transparency."

Plenck (2): "Videtur a vasis pellucidis in senio concrescentibus oriri."

Meckel (3): "It undoubtedly depends on the slowness with which the substance of the cornea is renewed, whence the fluids have more time to coagulate."

Walker (4): "It is composed of a firmer and more opaque substance than that of which the cornea is originally formed, arising from an impaired action of the secernent vessels which cease to pour out healthy corneal tissue."

Hays (5): "A deposit of lymph formed, perhaps, by a slow, chronic inflammation."

Seiler (6): "It arises from the approximation and closer attachment of the individual laminæ of the cornea, owing to the removal of the interstitial moisture."

Velpeau (7): "It cannot properly be attributed to disease of the cornea, the structure of that membrane not being altered nor its thickness increased."

Jeaffreson (8): "It is probably due to degeneration of the cornea itself, and not to any inflammatory deposit."

Lawrence (9): "No essential difference has been found in the part; there is no new growth nor any deficiency of its usual density. I can only compare it to the changes which take place in the internal coat of the arteries in old age."

Middlemore (10): "If it be not a mere alteration effected

by age. I am quite unprepared to say what it really is—no circumstance with which I am acquainted, except age, seems capable of producing it. The changes wrought in the arteries by age present the only analogy which now occurs to my mind."

Schön (11) "often examined the cornea affected with arcus, and found, the affected portion changed into a homogeneous—sometimes fat-like (speckartige) mass, often of considerable firmness. The laminated structure has entirely disappeared—the laminæ being consolidated into one mass: this was more particularly the case with the external than the internal laminæ."

Artha (12) states that the peripheral parts of the cornea are converted into a tissue analogous to that of the sclerotic, in consequence of an atrophy of its fibres incidental to old age, and likens the change to that seen under the microscope in a corneal nebula.

Canton (13) first accurately described the microscopic appearances. He removed the fresh cornea, cut it into four quadrants, and pinned each portion to a wax tablet, and allowed them to become dry in a current of air. After drying, a very thin shaving was removed from the edge of the preparation, placed upon a slide in dilute acetic acid, and covered. Canton's careful description (well illustrated in his book) (14) is as follows:—"With a power of 200 diameters, we shall observe myriads of fine oil-globules in the situation of, and constituting the arcus. Many larger globules are present, and this magnitude has doubtless been attained by the coalescence of the smaller spherules. We notice also that the oil-drops are arranged in two ways:—1st, in the direction of such of the lamellæ as have disappeared, and which they replace—a fatty degeneration or conversion—and, 2nd, in the interstices, or between the lamellæ—a fatty deposition. The slight elevation which the cornea presents at the site of the arcus is due to this latter condition.

"At the commencement of these changes, the oil-drops will be found in the corneal cells, and often, too, are present

in the fine branches or processes prolonged from these cells; in the same manner as the contents of the cellular elements of other organs are, primarily, affected where fatty degeneration occurs, and where, in the further progress of the affection, we may notice also the intercellular deposit." . . . .

"I have never seen Bowman's membrane (anterior elastic lamina) nor that of Descemet (posterior elastic lamina) to be otherwise than unaltered." . . . .

"With respect to the rim of cornea lying just external to the true arcus, and which, as a general rule, is unaffected, it will be not unfrequently found, in instances where an arcus is very broad, or a dense circle exists, that this part has become implicated also to a greater or less degree by a continuity of fatty degeneration from the arcus itself. This occurs more commonly above and below, than at the sides of the cornea." . . . .

"I have in some cases found the fatty degeneration to extend completely into the line of junction of cornea and sclerotic; and have occasionally seen the interstices of the sclerotic fibres as they are becoming corneal lamina, in many places, loaded with oil-drops."

Williams (15): "The arcus senilis owes its position on the upper segment of the cornea to the pressure of the upper eyelid, which, slight as it is, is sufficient to promote that change which results from weakened circulation."

His (16): "If thin sections through an arcus senilis are examined, the fat is principally found to consist of extremely fine globules, which are arranged in thickly set parallel thread-like rows. These parallel rows cross in various directions and so form a kind of thick network, which, when it is many layers thick, causes considerable opacity in the tissue. In some places larger groups of globules are collected, in the midst of which one or more stunted rudiments of a nucleus may occasionally be seen. These fat-drops have no capsule, and it is impossible to prove from their appearance only that they correspond to a degenerated corneal corpuscle. On the other hand there are undoubtedly

corneal cells with more or less fat within them, especially in the more posterior layers and in the parts where the opacity is less intense. Such cells are not actually enlarged, and the fat lies partly in the cell-body and partly in the fine processes and their ramifications."

Virchow (17) used the arcus senilis as a typical example of parenchymatous changes. He considered it a fatty degeneration of the corneal tissue, and due to deposition of fat in the cells themselves.

Castorani (18), in a paper to the Académie des Sciences, states:—(1) That the arcus senilis is the result of an immediate imbibition at the circumference of the cornea of the more or less abundant secretions of the conjunctiva; (2) that the more or less permanent contact of the eyelids with the cornea is an indispensable condition for this imbibition; (3) that the process of imbibition is in an inverted ratio to the resistance of the cornea and the density of the secreted fluids. The requisite conditions occur *par excellence* in the aged people who are subject to arcus, viz., increased conjunctival secretion, lessened mobility of the eyelids, and diminished resistance of the cornea.

Fuchs (19), in a paper on Pinguecula, says:—"Concretions of the same nature as those found in Pinguecula constitute the opacity of the cornea, which is called arcus senilis. Senescence of tissue leads therefore in the cornea to the same results as in the conjunctiva. . . . . The greenish-coloured round concretions which form it, lie for the most part immediately under Bowman's membrane. They consist of various sizes, from the smallest, scarcely visible up to 0·03 mm., mostly arranged in a single layer under Bowman's membrane. The larger concretions, by pressure on the posterior surface of Bowman's membrane, dig as it were niches in it, the largest thin the membrane very markedly and push it forwards. The concretions do not occur, however, only under Bowman's membrane; there are some which lie considerably deeper, between the corneal lamellæ. In some cases, indeed, I have only found large groups of

concretions here, whilst there were none present immediately under Bowman's membrane. On the other hand, there are concretions which are found within Bowman's membrane itself or even upon it. In the first case, when the concretions are very fine, the membrane appears as if dusted over, but it may also contain larger concretions. In the second case the concretions are imbedded between the cells of the corneal epithelium. There is never any relation to be perceived between the concretions and the cells of the corneal tissue ; the hyaline substance is here, exactly like the yellow masses in Pinguecula, lying separated and free upon the surface of the connective-tissue bundles (here corneal lamellæ). The concretions in the cornea give the same reactions as the concretions in Pinguecula. I may here only further state that they undergo no change whatever with ether or chloroform, so that therefore they are certainly not fat as generally held."

In his text-book (20) Fuchs says:—"The arcus senilis is formed by the deposition of hyaline masses and minute particles of lime in the more superficial layers of the cornea, close to the limbus ; its cause is assumed to be senile atrophy of the limbus, with involution of a portion of the vascular loops contained in it."

In a recent paper on a rare form of Peripheral Sclerosis and Atrophy of the Cornea (21), only found in conjunction with arcus senilis, Fuchs repeats his opinion that the arcus is due to hyaline degeneration, again citing the staining-reactions, and especially the fact that it stains black with Weigert or Pal's method, though hyaline material more usually only stains brown. He points out some resemblance to "Bandkeratitis," which Leber ascribes to drying of the cornea, and discusses the question whether the arcus is not due to an abnormal amount of moisture.

Leber (22) is of opinion that in arcus senilis there is a combination of hyaline degeneration with a deposition of calcareous material. In one case he obtained the very definite calcium reaction with oxalic acid.

Tweedy (23) writes :—"According to some investigations

made by the writer in conjunction with Mr. E. T. Collins, in the laboratory of Moorfields Eye Hospital, the change in the cornea consists in the presence of fine, highly refractive molecules, distributed along the course of the lymphatic spaces and channels of the superficial layers of the periphery of the cornea near the loop-endings of the capillaries of the conjunctival and episcleral blood-vessels. The greater portion of these molecules are not fatty, as is generally supposed; for, unlike fat, they are neither blackened by osmic acid nor dissolved by ether. They probably arise from mucoid degeneration of the protoplasm within the lymphatic channels and spaces of the cornea, and to some extent of the fibrillæ themselves. A few doubtful, blackened, fatty molecules may be seen here and there in sections stained by osmic acid. The fibrillæ are slightly wrinkled, and are more loosely held together than natural, and the spaces between the laminæ are wider. In support of the non-fatty nature of arcus senilis, it may be stated that wounds of the cornea, whether through the opacity, or to its inner side, or to its outer, and whether surgical or traumatic, heal in a natural way."

Takayasu (24), working in Greeff's laboratory, used Sudan III as a test for fat, and arrived definitely at the conclusion that arcus senilis is a fatty degeneration.

In my experiments I have used Sudan III and an allied stain, Scharlach R. The results confirm the description of Takayasu, and I have extended the observations by employing the stains as chemical indicators of the presence or absence of the arcus after the action of various reagents.

When stained with either of the dyes, the smaller globules are coloured reddish-yellow, the larger ones deep orange. With Scharlach R the stain has a brick-red tinge. The eyes were hardened in formol, the corneæ, including the iris and ciliary body, being then removed by a circular incision through the sclerotic 2 or 3 mm. behind the corneoscleral margin. The corneæ were then divided into quadrants, washed in running water to remove the formol, immersed in gum until penetrated, and cut with the freezing

microtome. After being freed from gum by warm water, sections were placed in saturated solutions of the stain in 70 per cent. alcohol. Stronger solutions of alcohol dissolve out the globules. In Sudan III the sections are well stained in from 10 to 15 minutes: longer treatment leads to slight diffuse staining of the corneal tissues, but this is washed out in subsequent treatment with 70 per cent. alcohol. It is possibly due to a fatty moiety in more stable combination in the normal tissues, for traces of fatty material can be extracted from almost every tissue of the body. Scharlach R stains more slowly, but does not tend to diffuse staining, even after prolonged (24 hours or more) treatment, and is, therefore, a more reliable test of differentiation. The sections after staining are washed rapidly in 70 per cent. alcohol, a more prolonged differentiation being required in sections over-stained by Sudan III. They are then washed in water, and counter-stained with Mayer's hæmalum or Ehrlich's hæmatoxylin. This stains the nuclei of the corneal corpuscles, &c., and to some extent delimits the corneal fibres. Hæmalum is preferable, as it is a pure nuclear stain: it may precede the treatment with the fat stain. The sections are then treated with tap-water until the hæmatoxylin is a satisfactory violet, and are mounted in glycerin.

In sections so treated, globules of varying size are seen to pervade Bowman's membrane and the corneal lamellæ in the affected area. Those in Bowman's membrane are usually the smallest, so small, indeed, that a general diffuse granular stain is all that is seen without the assistance of an oil-immersion objective. A few minute, isolated, well-defined globules may be seen in some cases. The stain is densest at the termination of Bowman's membrane, and this is the cause of the very definite peripheral edge of the arcus, as seen clinically. Bowman's membrane ends at an appreciable distance from the apparent corneo-scleral margin, and this distance is occupied by the clearer cornea which is characteristic of arcus senilis. That it is not in reality as clear as the centre will be evident from subsequent remarks.

Towards the centre of the cornea the stain in Bowman's membrane very gradually fades off, the distance varying with the development of the arcus in the particular cornea. It always, however, extends farther centrally than the stain in the substantia propria.

In the substantia propria the globules are limited to the lamellæ and corneal corpuscles, and are not contained in the lymphatic spaces and channels. The droplets are usually larger than those in Bowman's membrane, but the finer punctate appearance is seen around the larger drops. The largest drops are from 5—10 μ in length, and about half the width, the long axis being in the direction of the lamellæ, *i.e.*, more or less concentric with the surface of the cornea. This is due doubtless to the conditions of tension of the tissues. The smaller globules also show a similar disposition to be arranged in layers in the same direction: they are invariably punctate or spherical.

Tangential sections demonstrate that minute drops are also contained in the cytoplasm of the corneal corpuscles, extending into the processes of the cells, and showing a polar arrangement near the nucleus, as pointed out by His and Virchow.

Immediately beneath Bowman's membrane the stain is limited to the same extent, but each succeeding lamella is affected a little farther towards the periphery, so that in transverse sections a kind of staircase arrangement is seen. The deeper layers are usually less affected towards the centre of the cornea, and the density of the deposit also diminishes from before backwards, so that the middle layers of the substantia propria are little stained or not at all. Even under these circumstances, however, there is a further increase in the deepest layers, but the stain is not here so uniformly distributed, with the single exception that it is invariably extremely dense upon the surface of Descemet's membrane. With a low power, and particularly in thick sections, Descemet's membrane seems to be much involved, but with a higher power and in thin sections it is obvious

that it escapes entirely in reality, the dense deposit being only upon the anterior surface.

It is therefore obvious that the clearer peripheral ring of cornea is not entirely unaffected, but is comparatively clear owing to the absence of Bowman's membrane and the slighter implication of the more superficial layers of the substantia propria due to the staircase arrangement.

Beyond the true corneal margin there is a limiting area of sclerotic which I have never seen affected. Beyond this, however, there is frequently some deposition of fatty material in the deeper layers of the sclerotic.

Slight deposition of fat is almost invariably seen in the roots of the ciliary processes. This is probably physiological.

Using Sudan III or Scharlach R as indicators, it is found that the material is insoluble in water, dilute acetic acid, Müller's, Marchi's, Flemming's, and Hermann's solutions, &c., and that it is soluble in absolute alcohol, ether, chloroform, xylol, &c. Thus, after staining with Sudan III and immersion for half a minute in absolute alcohol, scarcely a trace of the stain remained, no globules could be seen, and the lamellæ had the appearance of normal lamellæ. Similar treatment for half a minute in ether resulted in total disappearance of the stain and the arcus. Hence arcus senilis cannot be demonstrated in paraffin or celloidin sections.

The globules can be dissolved out and restained upon the slide. Thus, a section was stained with Sudan III and mounted in 70 per cent. alcohol, covered with a cover-glass, and watched under the microscope. A small quantity of absolute alcohol was placed upon one side of the cover-glass and drawn through by means of blotting-paper. This was followed by a small dose of ether. The stain was rapidly dissolved out. The section was then restained with Sudan III upon the slide, the ether having evaporated. Many oil-globules were seen hanging about the anterior surface of the epithelium, well stained by the dye.

Fresh sections were stained by various solutions con-

taining osmic acid, for varying lengths of time. No definite staining was obtained with 2 per cent. osmic acid, unless it was allowed to gradually dry, in which case the whole section became black. It is known that osmic acid acts best after previous treatment of the sections with hardening reagents—2 per cent. osmic acid, after treatment of the section with 70 per cent. alcohol, failed to stain the deposit. Sections were placed in large quantities of Marchi's solution, Flemming's solution, and Hermann's solution, and examined at intervals from 24 hours to 3 weeks, being mounted in glycerin. In no case was there any true reduction of the osmic acid. The neighbourhood of the arcus certainly showed a blackish appearance, but this was apparently due to a difference in the relative refractility of the globules and the surrounding tissues, for under a high power it was seen that the blackness disappeared on slight alterations of focus. Further, after such treatment the arcus readily dissolved out when treated with ether, leaving no trace behind; and the blackened sections, when mounted in Canada balsam in the usual manner, could not have been distinguished from normal corneæ. There can, therefore, have been no true reduction of the osmic acid.

Fresh sections were treated by Bolton's modification of the Weigert-Pal method (25); *i.e.*, they were placed in 1 per cent. osmic acid for a few minutes, then in ammonium molybdate solution overnight, stained black with Kultschitzky's hæmatoxylin, and differentiated in Pal's solution. By this means the arcus was stained black. The stain was removed on treatment with ether.

Experiments were made to see if the globules contained phosphorus. For this purpose, Macallum's very accurate modification of Lilienfeld and Monti's method was used (26). Sections were immersed in ammonium molybdate solution for 24 hours in an incubator at 35° C.; they were then dipped in 4 per cent. solution of phenylhydrazin hydrochloride, washed in water, stained in Sudan III, and mounted in glycerin. The distribution of phosphorus in the

tissues was admirably shown. The arcus showed no trace, but was well stained by the Sudan III. Sections similarly treated, but not counter-stained with Sudan III, also showed the absence of phosphorus from the globules.

On the hypothesis that the material was fatty, an attempt was made to saponify the fat upon the slide. The section was mounted, without covering, in 70 per cent. alcohol. A small piece of metallic sodium was then placed in the fluid near the section and the reaction watched under the microscope. By this means sodium ethylate ($C_2H_5 \cdot ONa$), with some sodium hydrate (NaOH), was produced. The former is capable of saponifying fats in the cold. A large crop of acicular crystals (? $C_2H_5 \cdot ONa$) was formed, and the globules of the arcus disappeared.

Fresh sections stained with Lugol's solution (iodine dissolved in 1 per cent. solution of potassium iodide) stained the globules yellow, as is the case with most tissues. There was no trace of amyloid reaction.

Remarks.—Canton (27) states that Bowman's membrane is unaltered. This is inaccurate: as shown above, it is the main cause of the densest part of the arcus senilis.

He further states (28) that there is a slight elevation of the cornea at the site of the arcus. This is also inaccurate: but there is an undoubted, though usually slight, diminution in the thickness of the cornea peripheral to the arcus. The contrast probably led to the error. This slight thinning of the cornea outside the arcus points to a condition of sclerosis as a constant accompaniment of the degeneration. If this be so, it is interesting in connection with those rare cases of Peripheral Sclerosis and Peripheral Atrophy (Randsclerose and Randatrophie) which Fuchs (29) has recently described, and which only occur in association with a previously established arcus senilis. In these cases there is marked thinning of the cornea outside the arcus, which may be localised or may involve the whole ring round the cornea. It sometimes leads to ectasia, and consequent axial myopia. It is not usually preceded by inflammation, and is not an

ulceration, but a true sclerosis. It is to be distinguished from transient "Delle," which are described at the periphery, and also from Schmidt-Rimpler's (30) chronic peripheral "Furchenkeratitis," which is central to the arcus. In one of the four cases described by Fuchs, one eye was removed for glaucoma, and was examined microscopically. The sclerosed area formed a complete gutter round the cornea, which was ectatic. Bowman's membrane ceased 2 mm. from the edge, which is perpendicular on the corneal side and sloping on the scleral side. The gutter was 0·55 mm. wide, and the cornea at this part only 0·57 mm. thick. Outside Bowman's membrane the tissue was a looser connective tissue containing blood-vessels, there being a gradual transition from the lamellæ into this tissue. The epithelium was continuous over the surface, being of corneal type to the central edge and conjunctival over the floor, and thicker there owing to protection from pressure. Descemet's membrane was normal. Fuchs speaks of the appearances as indicating a "vorgeschobene Limbus." The same appearance of an invasion of the cornea by the conjunctiva of the limbus, leading to absorption of Bowman's membrane, strikes one when examining an ordinary arcus senilis, but I have been unable to convince myself that Bowman's membrane ends more centrally in these cases than normally. It is difficult, even under the microscope, to accurately fix the limits of cornea and sclera, and the only constant point to measure from is the spot where Descemet's membrane splits up at the ligamentum pectinatum iridis. Measured from this spot, Bowman's membrane does not end appreciably nearer the centre than in the normal eye.

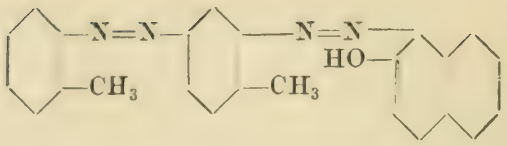
Canton (31) also refers to an encroachment of the conjunctiva upon the cornea, and states that it is greatest at the upper part. He quotes Wharton Jones's (32) accurate observation that the conjunctiva here "appears in its independent form, with its corion fully developed." He also divides the arcus into two parts—"the outer having a grayish-white or dusky tint, the inner one being milky in

colour. These are separated from each other by a clear, unaffected line of cornea, and through which the iris can be distinctly seen." He further quotes Mackenzie (33) as referring to this; and the observation is to some extent substantiated by the deep-lying affection of the sclerotic in Sudan-stained sections.

Canton's illustrations give very accurate representations of what is seen in unstained sections under the microscope, with the exception of the first, in which he depicts the arcus as a pyramidal opacity with the apex downwards, and Bowman's membrane, unaltered, extending far beyond it peripherally.

It is difficult to reconcile my observations with those of Fuchs and Leber, but in all probability they examined later conditions of the affection, or cases in which other degenerative changes were present, masking the true arcus, which would have disappeared in the specimens mounted in Canada balsam.

The conclusions arrived at by Mr. Tweedy and Mr. Treacher Collins are chiefly explicable by the behaviour of the globules to osmic acid. Even Takayasu, whose results I confirm for the most part, states that the globules stain with osmic acid, though with difficulty. I agree that they become blacker, but this is no evidence of a true fat stain, for I have proved that the osmic acid is not reduced. This result introduces a complication into the explanation of the action of Sudan III and Scharlach R. It has been shown by Altmann and others (34) that osmic acid only stains, *i.e.*, is only reduced by fats which are glycerides of the chemically unsatisfied acrylic series of acids ($C_nH_{2n-1}\cdot COOH$), of which olein is the commonest representative in animal tissues. Sudan III was first used by Daddi (35), the idea of its utility for microscopic purposes being derived from the fact that it was used commercially for staining fatty preparations, *e.g.*, pomades, &c. The nature of the dye and of its congener Scharlach R, which is chemically azo-orthotoluol azo-β-naphthol—



(Sudan III having the same formula, except that the two H's have not been replaced by (CH_3) groups) has been worked out by Michaelis (36). He explains their action upon the same principle as that of osmic acid, viz., that reduction of the stain is brought about by the action of fatty acids of the acrylic series. If this is true we should expect osmic acid, Sudan III, and Scharlach R to invariably act in the same manner. This is not the case with arcus senilis, and we are forced to the conclusion, either that there is some flaw in the chemistry of the action of these stains, or what is more probable, that osmic acid is more stable than the aromatic stains, which are reduced by a smaller amount of, or by less potent members of, the acrylic series.

It was thought possible, on account of the common occurrence of cholesterin in senile degenerations, that the fat-like material might be a cholesterin ester, or ethereal salt of a fatty or other acid. These bodies occur in large quantities in lanoline. The supposition was improbable *à priori*, for most cholesterin esters are insoluble in alcohol. On saponification they are split into the acid and cholesterin, which is an alcohol with the formula $C_{27}H_{45}OH$. It was just possible, if they were present, that cholesterin would crystallise out in its typical form upon the slide, when the section was treated with sodium ethylate. This, however, did not occur.

Most fats are not readily soluble in alcohol, and the globules of the arcus senilis show less solubility in it than in ether, but probably more than most fats.

In conclusion it may be stated that the arcus senilis is probably a fatty degeneration of the substantia propria of the cornea, the exact chemical nature of the fatty material being still undetermined.

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A REPORT OF THE CASES IN WHICH HAAB'S MAGNET WAS USED FOR THE EXTRACTION OF FOREIGN BODIES FROM THE EYE AT THE ROYAL LONDON OPHTHALMIC HOSPITAL.

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In the following paper all the cases have been reported in which foreign bodies were removed from the eye, directly or indirectly by Haab's magnet, up to the end of July, 1901. The cases which came under the care of Mr. Holmes Spicer have already been collected and published elsewhere, but it has been thought well to include all the cases in order that a complete report might be given:—

Case 1.—George S. was admitted under the care of Mr. Tay at Moorfields on January 10, 1900. His L. eye had been penetrated by a piece of steel 5 hours before admission. There was found to be a small wound at the inner part of the cornea, and a bead of vitreous was oozing from it. The iris was entangled in the wound. Through a small hole in the iris the opaque lens could be seen. On the following day Haab's magnet was applied by Mr. Collins; this brought the f. b. forwards, presumably from the posterior part of the vitreous, and caused the iris to bulge anteriorly. An incision was made through the sclerotic on the nasal side of the globe behind the ciliary body; the terminal of the small magnet was inserted, and the f. b. was withdrawn.

About a year after the accident there was found to be aphakia. A thickened capsule remained, which was somewhat adherent to the iris; this capsule was needled. With a correcting glass he now gets 6/6 and J. 1.

If in this case localisation by means of X-rays had been carried out, it is probable the f. b. might have been drawn through the opaque lens into the a. c. by Haab's magnet without any necessity for the use of the small magnet.

Case 2.—William R., æt. 28. A piece of steel penetrated L. globe 4 weeks previous to admission. He had attended another ophthalmic hospital, where a small magnet had been tried unsuccessfully, and where excision had been recommended. When seen—December 20, 1899—there was a corneal wound to the inner side, and a nebula just above the centre of the cornea; there were also definite signs of iritis. The lens was clear; there were vitreous opacities. V. L. = fingers at 4 feet. The tension was diminished.

On introducing the patient to Haab's magnet, the f. b. was drawn just over the pupillary border of the iris, but fell back again. Owing to failure of the current the small magnet could not be used, and further operation was deferred. Later, the patient was X-rayed, and the f. b. localised at the back of the vitreous. Haab's magnet being applied by Mr. Holmes Spicer—January 13, 1900—the f. b. was drawn into the a. c. After a corneal section had been made with a keratome, the small magnet withdrew the particle. Some lens matter was curetted; it was also necessary to perform a small iridectomy.

The patient was seen on January 20 of this year. He was found to have a thick capsular membrane, through which the details of the fundus could not be seen. Projection was perfect.

V. L. $\bar{c}$ sph. + 10 = 6/60. Field good.

Case 3.—George B. came to the hospital on May 10, 1901, and was seen by Mr. Flemming in the O. P. Department. He had been chipping a steel bar that morning, when a small piece of metal flew into his L. eye. There was found to be a small corneal wound situated between the centre of the cornea and the highest part of the corneal periphery, there was much injection, there was iritis, and the lens was wounded. The patient was at once taken to the theatre, and Haab's magnet applied. The f. b. was drawn through the lens and through the upper part of the undilated pupil; the iris prolapsed slightly after the exit of the f. b. through the wound, and the prolapse was snipped off. A good deal of inflammation succeeded the operation, and the lens vegetated through a rent in the anterior capsule. In a few days tension became normal, and the eye quieted down. The man was then discharged from the hospital, and kept under

observation in the O. P. Department. On June 16 he was re-admitted into the hospital, as the lens had become broken up to some extent, and it became necessary to remove it. This was accordingly done by curette evacuation. The eye again became quiet, and he was discharged. On July 2 the eye was irritable, and there was some injection; there was also some irritation of the unwounded eye; he was therefore re-admitted for the detachment of an anterior synechia which had formed. At this operation the capsular membrane, which was left after the curette evacuation, was needled, and a free opening was made. The patient was seen again on September 10, 1901, and he was found to read, with correction for his aphakic condition, 6/6 and J. 1.

Case 4.—Charles S., æt. 30, was struck in the L. eye by a chip of steel from the face of a hammer, 14 days before coming to the hospital. When seen by Mr. Holmes Spicer there was much conjunctival injection. In the upper and outer quadrant of the corneo-scleral margin was a wound in which the iris was entangled. The lens was clear. There was much hæmorrhage into the vitreous. V. L. = 6/36. At the outer canthus, beneath the conjunctiva, a small swelling could be seen. The skiagraph, which was taken, localised the f. b. in the region of the swelling about 6 mm. deep. On the day after his admission, May 12, 1900, Haab's magnet was applied to the globe without causing any pain; when applied to the outer canthus the patient experienced pain at once, and the conjunctiva bulged forward. The conjunctiva was divided over the region of the swelling, and extraction with the small magnet was attempted without success. On again applying Haab's magnet the foreign body appeared between the lips of the conjunctival wound, and was extracted with forceps. He was discharged from the hospital on May 21. V. L. = 6/60, when the condition was:—Fundus obscured by vitreous hæmorrhages; iris still somewhat adherent to the corneal wound. The patient was seen on February 23, 1901, when V. was found to be 6/6, partly. There was a large scar in the outermost periphery of the fundus. In this case the f. b. must have passed through the corneo-scleral ring, the ciliary body, the vitreous, and the sclerotic.

Size of f. b. = 8·5 mm. × 5·5 mm. × 3 mm.

Case 5.—On May 20, 1901, Henry S. was admitted into the hospital. He had been struck in the R. eye 14 days ago by a piece of steel from the face of a hammer. There was considerable injection, and a small wound in the lower part of the cornea; a small hypopyon was present; there was iritis, and some posterior synechiæ externally. The lens was completely opaque. V. = hand movements. The patient had been X-rayed while an O. P., and a spicule of steel, 3 mm. long, had been detected in the lens behind the iris in the situation where it was adherent to the lens capsule. With a needle Mr. Flemming tore through the anterior capsule of the lens. This manœuvre brought the f. b. into view; a peripheral corneal incision was made, and a terminal of the small magnet was introduced into the a. c. This, however, failed to remove the f. b. The patient was then introduced to Haab's magnet, which withdrew the f. b. without difficulty. The lens matter, which was found to be fairly fluid, was removed by curette evacuation. The patient was discharged after a fortnight with the eye quiet; the pupil was fairly dilated, and there was some soft matter in the pupillary area. Six weeks after the operation, after correction for the aphakic condition, he was able to read 6/9 and J. 1. His field, as taken by the perimeter, was normal.

Case 6.—Wm. B., æt. 42, first came to the hospital on September 9, 1897, having been struck in his L. eye with a piece of steel from a tool 10 weeks before. There was a scar on the cornea, the lower part of the lens was opaque, no details of the fundus could be seen. The eye remained quiet, and he was watched for a period of three months; no operation was thought advisable, as there was no evidence of the presence of a foreign body in the eye, and he ceased to attend. He returned to the hospital in September, 1899. On examination the eye was quiet; the lens was partly opaque; a red reflex was obtained from the upper part of the fundus, but no foreign body was seen. On November 3, the f. b. was localised by the aid of X-rays in the globe, situated on a horizontal plane, running 1 mm. below the lower margin of the cornea, and 7 mm. back from the plane of the centre of the cornea, almost in the middle line. On November 5, the condition of the eye was as follows:

Some general conjunctivitis; no visible wound; pupil irregular but active; lens opaque; no red fundus reflex. The patient was under the care of Mr. Holmes Spicer. On introducing the patient to Haab's magnet the f. b. was drawn into the a. c. and removed by the small magnet, after a corneal section had been made. The patient was discharged at the end of a week with the eye in a perfectly quiescent condition. On December 19 he was readmitted. The pupil was irregular, but fairly active. The lens had absorbed and a dense capsular membrane in the pupillary area remained. No details of the fundus could be seen.

V. L. $\bar{c}$ sph. + 10 = 6/60 T. = normal. Projection good. The capsule was needled, and a good central opening made. The patient continued to attend as an out-patient till February 10, 1900, when his V. L. was $\bar{c}$ + 13 D = 6/9.

Case 7.—Thos. T., æt. 27, on May 23, 1900, the L. globe was penetrated by a chip of steel from a lathe. He had had very little pain or inconvenience since the accident. When seen at Moorfields on July 1, by Mr. Holmes Spicer, he was found to have a corneal scar, and a traumatic cataract. The pupil was dilated under atropine, and the eye was fairly quiet. No red reflex was obtained from fundus. V. L. = hand movements. A f. b. was localised by the aid of X-rays in the vitreous 1·3 c.m. behind the centre of the cornea.

On July 4, the extraction of the f. b. was attempted. The patient's head being put on a level with, and at a distance of a few feet from Haab's magnet—the electric current was switched on gradually. At the same time the patient's eye was approached to the terminal, and he at once experienced pain. The f. b. was seen to come forward through the lens, and to take up a position on the lower pupillary border of the iris.

The patient, who had previously been sitting on a stool in front of the magnet, was transferred to the operating table. At this point the foreign body disappeared from view. An incision was made with a keratome at the lowest part of the corneo-scleral ring. The curved terminal of the horse-shoe magnet was introduced into the a. c., and the f. b. was drawn over the pupillary border of the iris, and removed. Some lens matter was evacuated

with a curette; a little infiltrated vitreous was also removed. On the patient's discharge, July 15, the eye was quite quiet, and the remainder of the lens matter was becoming absorbed.

On December 10, Dr. Edgar Stephenson, of Liverpool, under whose care the patient originally was, most kindly sent me a note as to his condition. The pupil was quite clear; the lens matter was all absorbed, but there were some capsular remains in the periphery.

$$V\ \bar{c}\ \text{sph.} + 11\ D = 6/9.$$
$$\bar{c}\ \text{sph.} + 15\ D = J.\ 1.$$

The patient had been back at work for some months, and has had no trouble of any kind with his eye.

Size of f. b. = (6·5 × 2 × 1) mm.

Case 8.—Manning H. was struck in R. E. by a chip when using hammer and chisel on April 30, 1901. He came to the hospital on May 4, and was admitted under the care of Mr. Lang. The eye was quiet; there was a small corneal wound in the upper and inner quadrant of the cornea near its margin. There was no iritis, the lens was opaque. On May 8 the f. b. was localised accurately by the X-rays. It was found to be behind the lens on its nasal side, about the level of the lower border. The pupil was dilated with atropine but full mydriasis was not obtained. The f. b. was drawn forwards from its position by Haab's magnet to the lower and inner quadrant of the iris where a bulging was produced. All attempts to disengage the f. b. from the iris and bring it into the pupillary area failed during the use both of Haab's and the small magnet. The patient was placed upon the table and a peripheral incision was made with the keratome below, at the corneo-scleral junction. Iris and f. b. at once prolapsed. The f. b. was removed with the small magnet and a small iridectomy was done. On the 15th June, with a low astigmatic correction, the patient read 6/6 and J. 1. The condition of the eye was as follows:—The iris was adherent below to the keratome wound, there was some slight lenticular opacity—and very fine vitreous opacities. The patient complained of some asthenopia when reading.

Case 9.—Thomas C., æt. 47, was admitted to Moorfields under the care of Mr. Treacher Collins, on May 14, 1900.

Three weeks previously a steel splinter had penetrated the L. eye while chipping brickwork. He had pain at the time and this was getting worse. Many years previously he had had an injury to the same eye which left a small scar in the sclerotic, at the inner side of the cornea. On examination he was found to have some injection of the conjunctiva. There was a wound in the upper hemisphere of the cornea. Corresponding to this was a notch on the edge of the iris. V. L. = 6/60. The patient was introduced to Haab's magnet, which caused the f. b. to traverse the lens and bulge forward the iris. This occurred after several separate trials of the magnet.

A corneo-scleral incision was made with a keratome, and an attempt was made to extract the f. b. with the horse-shoe magnet. This was unsuccessful because the piece of steel had fallen down behind the iris and was not in the a. c. The patient was then anæsthetised, an iridectomy was done, and the f. b. was removed with the small magnet. As a result of the operation a traumatic cataract developed. He was admitted for the extraction of this on October 17, 1900; this was done, with iridectomy, and in due course he was discharged from the hospital. I saw him again at the end of January of last year; in the pupillary area there was fine capsule, to which the iris was adherent in places, the fundus was healthy, the field was good. The refraction, as obtained by retinoscopy, was + 18 D.

V. L. $\bar{c}$ sph. + 17 D = 6/12. $\bar{c}$ sph + 21 D = J. 1.

In this case the f. b. was not located by X-rays. Had this been done injury to the lens might have been avoided.

Case 10.—Edward G. was admitted under the care of Mr. Collins, on August 29, 1901. He had been wounded in the R. E. by a chip of metal from a steel hammer a week previously. There was a wound in the ciliary region, and hæmorrhage into vitreous and a. c. It was not known whether the f. b. was present or not.

The patient was introduced to Haab's magnet and the sclerotic was seen to bulge slightly in the neighbourhood of the old wound. An incision was made through the sclerotic at this point and the f. b. was removed by the small magnet.

A little vitreous was lost.

I saw the patient a month after the operation; the lens was clear, and V. = 6/12. In this case, although the actual removal of the f. b. was effected by the small magnet, Haab's magnet played the chief part. The f. b. previous to operation occupied an unknown position in the vitreous, whence it was drawn into such a situation that the use of the small magnet became possible (after an incision in the sclerotic had been made) without disturbing the vitreous by its terminal. In fact, before Haab's magnet was applied it was not actually known that any f. b. was present in the globe.

CASE 11.—Wm. N., æt. 16, under the care of Mr. Holmes Spicer. On March 16, 1900, a piece of steel flew into the patient's L. E. from a chisel. He was found to have a scar at the outer part of the cornea, a hypopyon, and iritis. Both cornea and lens had been wounded; there was a dull red fundus reflex only; projection was good. V. = hand movements.

On March 20 the hypopyon had disappeared. The f. b. was localised in the vitreous, 1·2 cm. behind the centre of the cornea, and 4 mm. to temporal side. A few days later the patient was introduced to Haab's magnet, and the f. b. was drawn (without further wound of the lens) into the a. c. through the lower periphery of the iris. Probably it came through the zonule. A section was made with a keratome, and on inserting the curved terminal of the horse-shoe magnet, the f. b. adhered to it, and was removed. When the patient was seen eight months later the condition was as follows:—An anterior synechia at the site of the corneal wound; an active pupil, and some anterior and posterior capsular opacity of the lens. After correction of hypermetropia patient could read 6/12 partly.

The patient now follows his trade of carpenter with ease.

CASE 12.—Fredk. C. was admitted to Moorfields under the care of Mr. Tay, in September, 1900. He said that two days previously he had been struck in R. eye by a piece of metal. The L. eye had been excised 10 years ago, at Moorfields, as the result of an accident. On examination there was found to be a hypopyon and iritis. The f. b. was seen to be lying on the iris. There was a wound in the lower part of the cornea. The small magnet was tried unsuccessfully by Mr. Lawson, the terminal

being inserted through the corneal wound. The patient was then introduced to Haab's magnet. By its attraction the f. b. was caused to emerge slowly from the corneal wound. The patient was discharged from the hospital on October 10. Vision was then 6/18 after correcting some astigmatism.

I saw the patient on January 31, 1901. He complained of pain at night, and during the day he had sensations of "smoke" in his eye. He was led about by his wife and professed to be entirely unable to find his way about alone, although he avoided obstacles when the guiding hand was removed. The V. was 6/36 slightly improved with correcting glass. Vision of this degree would certainly enable the possessor to steer clear of large obstacles when walking about. There was very slight opacity of the lens, but the fundus could be distinctly seen. It appeared to be healthy except for a few spots at the macular region—of lighter colour than the surrounding fundus—probably significant of colloid degeneration. His field of vision was taken and proved to be remarkably small. It was entirely included in the 20 mm. circle of the perimeter chart. It appeared that the patient was making a claim against his employers for the damage he sustained by his accident.

From what has been said above one conjectured that the man was a malingerer, especially as on the next occasion when he was seen—about a month later—the vision had improved to 6/24 with the same correction, although the field was even smaller than before. The tension was normal.

Case 13.—Alice C., æt. 97, was admitted under the care of Mr. Morton, August 2, 1900. Three years previously a piece of steel had penetrated R. E. For about a year before her admission she had had some inflammation in the injured eye, but no affection of the other. On examination there was found to be a scar in the outer part of the cornea, with a small piece of steel embedded, projecting into the a. c. and touching the iris. The pupil was semi-dilated. There was distinct optic neuritis. V. = 6/24.

The patient was introduced to Haab's magnet, and the f. b. was disentangled from its adhesion to the cornea and iris, and

was attracted into a different position in the a. c. so that it lay free. An incision was made in the cornea and the f. b. was withdrawn by the small magnet. A small iridectomy was performed as there was some adhesion of iris to the wound. The patient was discharged from the hospital on August 13, with the eye quiet.

She was seen at the end of October, when there was apparent paralysis of the R. external rectus; there was also an anterior synechia. On November 12, the squint, previously so marked, had disappeared. There was no optic neuritis now present —V $\bar{c}$ hypermetropic correction = 6/18.

Case 14.—Wm. L. was seen by Dr. Rayner Batten on May 14, 1900, to whom I am indebted for these notes. He said that three days previously he had had a blow on the L. E. with a chip of metal from a chisel. A linear wound of the cornea was present, with a corresponding wound of the iris beneath. The lens was partially opaque, V = P. L. T = −1. On May 15 there was a small hypopyon and iritis; on this day Dr. Rayner Batten brought the patient to Moorfields, where the f. b. was localised in the vitreous by X-rays. The patient was introduced to Haab's magnet by Mr. Lang, and after several trials the iris bulged forward at its lowest part. An unsuccessful attempt was made to draw the f. b. over the iris into the a. c. The patient was then anæsthetised. After a corneal section had been made an iridectomy was done, and the f. b. was removed with the small magnet. Some opaque fibrous-looking tissue was removed at the same time, which appeared to have encapsuled the f. b.

On the 24th of the same month the patient was discharged from Moorfields, and returned to Dr. Batten's care. On February 11, 1901, Dr. Batten, at the Western Ophthalmic Hospital, removed the opaque lens.

On March 7, V $\bar{c}$ correction was found to be 6/36. There was thickened capsule in the pupillary area.

It is to be noted that the patient had divergent strabismus, and that the injured eye was the non-fixing one.

Case 15.—Mark A. was admitted to Moorfields saying that he had had an accident with a piece of steel to his L. E. on the previous day. There was a wound of the sclerotic to the inner

side of the cornea; there was mydriasis, probably due to the use of atropine; inflammatory reaction was not particularly marked. On ophthalmoscopic examination a f. b. was seen lying in the vitreous with its long axis nearly vertical. The posterior edge seemed to be in close relation with the fundus, the details of which were obscured by vitreous opacity. V. L. = 3/60.

On July 27, 1900, the day after the patient's admission, an incision was made by Mr. Marshall through the sclerotic to the temporal side of the ciliary body. Several attempts were made to withdraw the f. b. by inserting the terminal of the small magnet, but these were unsuccessful.

Mr. Marshall then introduced the patient to Haab's magnet, which at once withdrew the chip of steel from the scleral wound. I saw the patient on January 26, 1901; his vision was then 6/36. There were some striæ in the lens, and some slight vitreous opacity. To the outer side of the disc was an area of old choroido-retinitis; this corresponded with the site of the f. b.

Case 16.—Albert P., under the care of Mr. Spicer, came to the out-patient department complaining that a piece of steel had struck his R. E. He thought that nothing remained in it. He said that the fragment was $\frac{1}{2}$ inch long, and had been found on the ground after the accident. On examination there was seen to be slight conjunctival injection, a vertical wound in the outer hemisphere of the cornea, and some iritis. The lens was becoming opaque. V. R. = Hand movements. The tension was diminished. He was treated as an out-patient until April 7. In the interval he was X-rayed, and the f. b. was localised in the vitreous, probably close to the retina. He was admitted to the hospital, and Haab's magnet being applied, the f. b. was drawn into the a. c. The patient was then anæsthetised: with a Graefe knife, a section was made upwards, and the f. b. was extracted with ease by the small magnet. Some lens matter escaped, and some was evacuated with the curette. Iris prolapsing, a small iridectomy was done. The patient was seen January 20, 1901; a shrunken, calcareous capsule was present, which almost entirely blocked the pupil. On either side was seen a faint red fundus reflex.

V. R. $\bar{e}$ sph. + 10 = 6/60.

Size of f. b. = (5·5 × 3 × 2) mm.

Case 17.—Henry J. was struck in L. E. by a chip of iron, October 18, 1900. On October 29 the patient was admitted to the hospital under the care of Mr. Gunn. There were no signs of inflammation. A scar was present in the upper part of the cornea, and corresponding to this there was a small hole in the iris near the pupillary border. Opacities were present in the posterior part of the lens and in the vitreous. An especially large opacity was seen in the uppermost part of the vitreous. This was supposed to be the f. b. V. = 3/60.

On October 31 Haab's magnet was applied, and the f. b. was drawn into the a. c. At the lower and inner periphery of the cornea a section was made with a keratome; through this incision the f. b. was withdrawn with difficulty, owing to its shape, by the small magnet.

On November 3rd, a skiagram showed no f. b. to be present. A week later the eye had quieted down perfectly. In the lowest part of the a. c. there was a small piece of lymph, which probably encapsuled the f. b. The hole in the iris was very apparent, the lenticular opacity had increased slightly. V. = 6/60.

Case 18.—Wm. K. was admitted to the hospital on November 16, 1900, under the care of Mr. Gunn. His R. E. was said to have been struck by a piece of metal six months previously. By the X-rays a piece of steel was localised in the R. eyeball. It was found to be lying vertically behind the posterior surface of the lens in the lower part of the vitreous. The eye was perfectly quiet. It was impossible to decide on the site of the original wound. The pupil was semi-dilated and inactive; the iris was yellowish in colour, but this was probably of no importance, since the irides were said to be heterochromic from birth. On the anterior capsule of the lens were many spots of reddish-yellow pigment. The lens was partially opaque. V. = counted fingers at 1 metre. On November 17 Haab's magnet was applied, and drew the f. b. through the lens, until the iris bulged forwards at its lowest part. The patient was then anæsthetised; a corneal incision was made with the keratome, and the small magnet withdrew the f. b. A small portion of iris, in which the f. b. was entangled, prolapsed and was cut off. The patient was discharged from the hospital on November 22. The pigment on the anterior

surface of the lens had increased. No view of the fundus could be obtained, owing to the lenticular opacity, which was situated at the posterior pole for the most part. The patient was seen again on January 15, 1901, when the lens was opaque; on the anterior capsule the pigment previously seen was still present. Projection was good. Mr. Gunn thought that the pigment probably consisted of oxidised iron; he did not consider it advisable to carry out any operative procedure on the lens for some time.

Case 19.—Charles C. was admitted into the hospital on October 11, 1900, under the care of Mr. Morton, having been struck in the R. E. by a piece of steel from a hammer. He was treated at a provincial hospital for three weeks, and was then advised to come to Moorfields. On examination there was found to be slight ciliary redness, and signs of recent iritis. On the nasal side of the cornea was a small scar with a tag of iris adherent to it. The lens was opaque; V. = hand shadows; projection bad; T. full. The L. E. was normal. On the following day the anterior synechia was divided by Mr. Morton with a Lang's knife. The f. b. was localised by X-rays in the vitreous on the nasal side of the globe at the lowest part.

On October 16 Haab's magnet was applied, and drew the f. b. forwards until the lower part of the iris bulged forwards. The eye was then directed downwards, and the f. b. was drawn by the magnet through the opaque lens into the upper part of the a. c. On the current being broken the f. b. fell to the lowest part of the a. c. An incision was made in the lower part of the cornea, and the terminal of the small magnet was introduced. The f. b., however, lying in the pocket formed by the limbus below the level of the incision, could not be removed. An attempt made with the forceps to withdraw the f. b. also failed. The patient was then introduced to Haab's magnet again, and the f. b. was drawn through the incision which had been made. I saw the patient three months later, the pupil was then irregular, and there were many posterior synechiæ. There was a dense band of fibrous tissue running vertically across the pupillary area; a faint white reflex only was obtained from the fundus. V. = hand movements. Projection bad.

Case 20.—Fred. O. was admitted to the hospital on June 7,

1900, under the care of Mr. Morton. He said that his L. E. had been injured by a piece of steel about 6 months previously. He had had much inflammation at times, but the reason he came to the hospital was because V. L. was failing. On examination there was found to be a corneal scar over the inner and lower part of the pupil. There was a considerable amount of inflammation, as shown by the conjunctival injection, the keratitis punctata, and the muddiness of the iris, which were present. Granular opacities at the posterior pole of the lens, and floating opacities in the vitreous were also present. V. L. = 6/60 not improved.

A f. b. was localised by X-rays in the vitreous, just behind and below the lens in the mid-vertical line. On the following day the patient was introduced to Haab's magnet, and the f. b. came forwards carrying with it much of the opaque matter which previously had been seen at the posterior pole of the lens. When the current was interrupted the f. b. disappeared from view. The current was again applied, and the f. b. became lightly entangled in the pupillary border of the iris, partially projecting into the a. c. An incision was then made over the site of the f. b., not quite at the periphery of the cornea, and the f. b. was removed with the small magnet. A fold of iris being in close proximity to the wound was seized with forceps and snipped off with de Wecker's scissors.

After a few days there was a greyish reflex from the vitreous. On June 22 the patient was seen by Mr. Morton and Mr. Silcock in consultation, and excision was advised, on the grounds that the injured eye was not likely to be of service for visual purposes, and would probably become a source of danger to its fellow. The operation was, however, refused by the patient. He was therefore discharged from the hospital. On January 20, 1901, I saw the patient again, and found the eye to be fairly quiet. There was a posterior ring synechia, and the pupil was blocked by organised inflammatory exudate. As far as could be seen the lens was clear. There was only a dull reflex from the fundus. The patient was able to count fingers at 1 metre, and his projection was perfect. On April 29 the condition was less favourable, the iris on the left side was very different in colour to that on the right, though there was no actual inflammation

present. Projection was defective: since then I have not seen the patient.

Case 21.—William S. was admitted under the care of Mr. Morton on March 8, 1901. Six months previously his R. E. had been injured by a chip of metal. He had been X-rayed at a general hospital on several occasions, and the f. b. had been located in the globe. An attempt to extract it with the magnet had failed, and excision had been advised.

He then came to Moorfields; there was found to be a small coloboma of the iris up and in, and the iris was adherent to the iridectomy wound. There were posterior synechiæ and the lens was completely opaque. Projection good.

The f. b. was precisely localised in the lens by X-rays. The patient was introduced to Haab's magnet, and on making the current the iris bulged at its lower and inner quadrant. The eye was directed downwards, and a f. b. of large size appeared in the pupillary area. On breaking the current, it partially slipped back behind the iris. The patient was then placed on the table, and a large horizontal incision was made almost in the centre of the cornea. Haab's magnet was applied behind the patient's head while he was on the table, and the f. b. was removed. In size it was 3 × 2 × 1·5 mm. With the f. b. was withdrawn infiltrated vitreous and lens matter. Protruding vitreous and lens matter was snipped off. The eye quieted down, but, in spite of the use of mydriatics, the pupil became small, and the iris contracted adhesions with the capsule of the opaque lens. I have been unable to communicate with the patient to ascertain his present condition, but as nothing further has been heard of him we may suppose that the eye is still quiescent.

Case 22.—Harry H. was admitted into the hospital under the care of Mr. Tay, March 20, 1900, with a history of a scale from an anvil at which he had been working, striking R. E. on previous day. The same eye had been injured in 1886, and the traumatic cataract which resulted had been removed by needling and curette evacuation. On admission the f. b. was seen in the lowest part of the a. c.; the wound of entrance was situated in

the cornea above and to the inner side of the present site of the f. b.

The patient was at once taken to the theatre, and the f. b. was drawn into the upper part of the a. c. without difficulty by Haab's magnet. With a keratome an incision was made at the outer margin of the cornea, and the f. b. was removed with the small magnet. The patient was kept in the hospital for 6 or 7 hours, and was then sent out owing to lack of vacant beds.

He attended as an O. P., and convalescence was soon established.

On December 26, 1900, after an absence from the hospital of some duration, he came up and was found to have vesicular keratitis and increased tension in R. E.

On February 25, 1901, cornea was much clearer, and the pain which had previously been present was diminished.

V. = hand movements. Tn. Since then the patient has not been seen.

The following cases resulted in excision of the injured globe. They will therefore be considered as briefly as possible :—

Case 23.—Wm. M. (under the care of Mr. Lawford in 1899). Haab's magnet in this case drew the f. b. through the opaque lens into the a. c. It was removed thence *viâ* the original wound by the small magnet, and at the same time some lens matter was curetted. This operation was performed on the second day after the injury. Later a second curette evacuation was performed, and the eye was excised a few days afterwards in a condition of panophthalmitis.

Case 24.—(Under the care of Mr. Lawford in 1899.) At the time of operation in this case the aqueous was leaking through a corneal wound ; there was iritis, and the lens was opaque. Haab's magnet was used to draw the f. b. into the upper part of the a. c., and the small magnet to extract it thence. A hypopyon developed, but inflammatory manifestations subsided in a few days, and the case seemed to be going on well. After some days there was found to be no P. L., and the eyeball was accordingly removed. When the globe was examined, the vitreous was found to be shrunken and invaded by a purulent exudate.

CASE 25.—(Wm. C., under the care of Mr. Marshall.) Here the injury occurred two days before the patient came to the hospital. There was considerable inflammation, and the lens was opaque. Haab's magnet withdrew the f. b., and iris prolapsing in its wake an iridectomy was performed. Two days after the operation the projection of light was defective and the tension was low. On the day after the operation, the patient insisted on being discharged from the hospital. He returned the next day in pain, and Mr. Marshall excised the eye. On examining the globe the vitreous contained a stringy band running from the ciliary body below to the original site of the f. b. in the retina. The retina was not detached.

CASE 26.—(Chas. P., Mr. Fisher.) The patient's eye had been injured 4 days before the date of extraction of f. b. by Haab's magnet. The f. b. was localised by the X-rays in the vitreous; by Haab's magnet it was drawn through the clear lens into the a. c. A peripheral corneal section was made with a keratome, and the small magnet was used to remove the f. b. thence. The eye quieted down fairly well, and the patient was discharged. Two months after the operation there was a little redness of the conjunctiva and epislera localised to the neighbourhood of an anterior synechia. The eye was soft, and vision was only hand movements. Accordingly Mr. Fisher advised excision, which was performed. On examining the globe the retina was found to be detached and to be adherent to the infiltrated track in the vitreous which marked the path of egress of the f.b.

CASE 27.—(Wm. W., Mr. Marshall.) In this case the injury occurred the day before admission. There was iritis and a traumatic cataract. He had never seen well with the eye. Haab's magnet removed the f. b. through the corneal wound. Ten days after operation the lens capsule and iris were found to be adherent to the wound. An iridectomy and a partial curette evacuation were done. Later it was found that the globe was shrinking, the lens capsule was still entangled in the wound, and vision was merely shadows. The eye was therefore excised. On examination the vitreous was found to be shrunken.

CASE 28.—(Wm. B., Mr. Morton.) The injury occurred the

day before the operation; there was iritis and a traumatic cataract. Haab's magnet removed the f. b. through the corneal wound. A fortnight after the operation injection persisted and a capsular synechia, which was present, was divided. A fortnight later an iridectomy was done, and an abortive attempt was made to divide the still present synechia. A month after the operation the eye was still much inflamed and was excised. On examination of the eye the vitreous was found to be infiltrated and shrunken, and the retina detached in places.

Case 29.—(David A., Mr. Collins.) Injury 2 days before admission when there was much iritis and a hypopyon. The pupil was blocked with lymph. The f. b. was localised in vitreous by X-rays. Haab's magnet drew the f. b. forwards until the iris was bulged. A small iridectomy was done and the small magnet was inserted through the wound without result. Haab's magnet then removed the f. b. A week after the operation there were the clinical signs of suppuration in the vitreous. Accordingly the contents of the sclerotic were eviscerated and a glass globe was inserted.

Case 30.—(Frank P., Mr. Spicer.) The injury occurred the day before the operation when there was slight conjunctival injection, a prolapse of iris through the corneal wound, and a traumatic cataract. Haab's magnet removed the f. b. and the prolapse of iris was snipped off. A little soft lens matter, which seemed to be mingled with vitreous, welling from the wound was cut off. A violent iritis followed the operation and the tension became raised. Some lens matter was removed by curette evacuation and the a. c. was washed out with saline solution, but in spite of this excision became necessary and was performed. On examination the retina was found to be detached, and under it there was a purulent exudate.

Case 31.—(Robert S., Mr. Marshall.) The injury occurred the day before operation. There was much injection, a patent corneal wound, and a traumatic cataract. Haab's magnet removed the f. b., which was followed by a bead of infiltrated vitreous. The injection which was present after the operation did not subside. At different times three operations were per-

formed without benefit. They were:—Needling, curette evacuation, and division of anterior synechia. Finally excision was performed. On examining the excised globe the vitreous was found to be shrunken and to contain a large blood clot.

Case 32.—(Herbert A., Mr. Marshall.) The injury occurred 5 days before operation. By X-rays a piece of steel $\frac{3}{4}$ inch long was located in the vitreous. This was removed by Haab's magnet through the original scleral wound which had to be enlarged for the purpose. As was to be expected excision became necessary later on.

Case 33.—(Wm. M., Mr. Spicer.) This patient came to the hospital with a history of injury to the R. E. by a piece of metal early on the same day. There was a corneal wound which had not closed. The iris was entangled in the wound and was in a state of acute inflammation. The lens was opaque. The patient was introduced to Haab's magnet the same day, which drew the f. b. into the a. c. Through the reopened wound the small magnet removed the particle.

Some days after the operation, which was followed by considerable hæmorrhage, the iris was found to be entangled in the wound. The eye was in an irritable condition, and was considered to be a source of danger to its fellow. Mules' evisceration operation was therefore performed.

Case 34.—(Charles G., Mr. Spicer.) This patient was admitted 2 days after the penetration of a piece of steel into his L. E. He had much conjunctival discharge; the cornea was hazy, and there was a small central wound. There was marked iritis.

The f. b. was removed on the same day by Haab's magnet by way of the original wound. On the following day, the inflammation having become more acute, the edges of the wound were touched with the galvano-cautery. The eye appeared likely to become a source of danger to its fellow, and Mules' operation was accordingly performed a week after the date of injury.

In the following cases Haab's magnet failed to remove the f. b.:—

Case 35.—Under Mr. Silcock in 1899. Here the injury was of 3 days' standing when the patient came to the hospital. There was a hypopyon and iritis. Haab's magnet caused great pain when the patient was introduced to it, but did not remove the f. b. The eye was excised, and the f. b. was found to be firmly entangled in the ciliary body.[1]

Case 36.—Joseph B. (under Mr. Gunn) was admitted November 29, 1900. About 3 months before his L. E. had been injured by a chip of metal. The lens was completely opaque and there were posterior synechiæ; the eye was quiet. Haab's magnet was tried but without other result than causing some pain. The small magnet was also tried without success. The eye was therefore excised, and in it a f. b. was found encapsuled in lymph and adherent to the ciliary body. It was in the position indicated by the X-rays localisation which had been previously carried out.

Case 37.—James N. (under the care of Mr. Collins) was injured by a piece of steel on the day of admission, March 9, 1901. There was intense chemosis, iris was prolapsed through corneal wound, and the lens was opaque. Haab's magnet was applied at once by the House Surgeon without result. Two days later the iris was freed by Mr. Collins and the small magnet was tried unsuccessfully. By X-rays a large piece of metal was found to be embedded at the back of the globe and to be projecting through the coats of the eye into the orbit. Haab's magnet when tried directly in front of the patient elicited no pain, but when the pole was placed in the temporal region the patient felt pain. The eye was excised and the f. b. was found in the position described in the X-ray report. Its size was:—
$14{\cdot}5 \times 11 \times 1{\cdot}5$ mm.

The damage done to the eye was such that the House Surgeon did not consider it worth while to remove the prolapsed iris, as he thought that excision would eventually be necessary.

Case 38.—Robert B. was admitted to the hospital on June 18, 1900. He had been struck in L. E. by a piece of steel when at work 3 weeks previously. He had been attended during this time by a country doctor. He then went to see

Dr. Johnson Taylor of Norwich, who sent him to Moorfields. The R. was a blind, shrunken eye. On admission the eye was found to be in a markedly inflamed condition. There was a small scar in the outer and lower part of the cornea. The a. c. was occupied by a small hypopyon. The pupil was semi-dilated by atropine. In the anterior part of the lens were some small opacities. By focal illumination an opaque yellowish mass could be seen in the vitreous; there was no red reflex. V. = hand movements. Projection defective. Tension diminished.

On the following day Haab's magnet was tried. Much pain was caused and the patient became collapsed. Further operation was therefore deferred.

By X-rays the f. b. was localised in the lower part of the vitreous, in the middle line behind and below the lens.

On June 22, the patient was anæsthetised. Haab's magnet was applied but without a successful result; there was considerable difficulties in applying the magnet, since its movement is rotary in a horizontal plane only, and it is, therefore, difficult to approximate the pole to the eye of a patient who is in a horizontal posture. Finally the f. b. was removed without difficulty after a scleral opening had been made. The eye became quiet and the patient was discharged from the hospital. Dr. Johnson Taylor saw the patient on January 18, 1901, and was kind enough to inform me as to the condition of the eye, viz.,—that the globe was shrunken, that the lens was opaque, and that there was no perception of light. Size of f. b. 2·5 × 2 × 1 mm.

In the last four cases it has been said that Haab's magnet failed to remove the piece of steel present in each case. There were, however, definite causes for this failure. In the first two cases the foreign body was encapsuled and adherent to the ciliary body. In the third case the piece of steel was very large, and was firmly wedged in a rent in the sclerotic. In the fourth case the difficulty in manipulating the magnet with the patient in a supine posture has been described. In every case the patient experienced pain during the application of the magnet. The sensation of pain is a constant occurrence when a patient, with a magnetisable particle in his eye, is brought up to Haab's magnet, and is of diagnostic

value, for if there is no pain, the eye may be supposed to contain no magnetisable particle.

There has been only one possible exception to this rule in the cases at Moorfields, and in this particular instance there was no proof that the f. b. was of magnetisable nature. The case was under the care of Mr. Collins. Ophthalmoscopically a f. b. could be detected near the retina, but Haab's magnet when applied was without effect. X-rays confirmed the presence of a f. b., and Haab's magnet, again applied, did not cause any pain.

Case 39.—Geo. G., under the care of Mr. Tay, was not seen until 2 days after the injury, when there was much chemosis of conjunctiva and a large hypopyon. There was no P. L. Haab's magnet removed a f. b. through a corneal incision. Excision was necessary later. Size of f. b. 2·5 × 1·25 × 0·5 mm.

The first 34 cases, in each of which Haab's magnet was instrumental in removing a f. b. from the eye, may be tabulated as follows:—

No.	Patient.	Surgeon.	X-rays.	Operations.	Further operations.
1	Geo. S. ..	Mr. Tay ..	No	Haab's magnet. Small magnet	Needling .. .
2	Wm. R. ..	Mr. Spicer ..	Yes	Haab's magnet. Small magnet. Curette evacuation. Iridectomy	.. ..
3	Geo. B. ..	Mr. Flemming	No	Haab's magnet. Iridectomy..	Curette evacuation Needling.
4	Chas. S. ..	Mr. Spicer ..	Yes	Haab's magnet	.. ..
5	Harry S. ..	Mr. Flemming	Yes	Haab's magnet	Curette evacuation
6	Wm. B. ..	Mr. Spicer ..	Yes	Haab's magnet. Small magnet	Needling .. ..
7	Thos. T. ..	Mr. Spicer ..	Yes	Haab's magnet. Small magnet Curette evacuation	.. ..
8	Manning H.	Mr. Lang ..	Yes	Haab's magnet. Small magnet. Iridectomy	.. ..
9	Thos. C. ..	Mr. Collins ..	No	Haab's magnet. Iridectomy. Small magnet	Extraction c̄ iridectomy.
10	Edwd. G. ..	Mr. Collins ..	No	Haab's magnet. Small magnet. Iridectomy	.. ..
11	Wm. Neve..	Mr. Spicer ..	Yes	Haab's magnet. Small magnet	.. ..
12	Fred. C. ..	Mr. Lawson ..	No	Haab's magnet..	.. ..
13	Alice C. ..	Mr. Morton ..	No	Haab's magnet. Small magnet. Iridectomy	.. ..
14	Wm. L. ..	Mr. Lang ..	Yes	Haab's magnet. Iridectomy. Small magnet	Curette evacuation
15	Mark A. ..	Mr. Marshall	No	Haab's magnet. Scleral incision	.. ..
16	Albert P. ..	Mr. Spicer ..	Yes	Haab's magnet. Small magnet. Curette evacuation. Iridectomy	.. ..
17	Henry J. ..	Mr. Gunn ..	No	Haab's magnet. Small magnet	.. ..

Site of f. b.	Size of f. b.	Vision.	Remarks.
Vitreous ..	.. ..	c̄ correction 6/6 J. 1..	Traumatic cataract absorbed spontaneously.
Vitreous ..	.. ..	c̄ correction 6/60	Patient is aphakic. There is a thick capsular membrane. Projection and field good.
Vitreous ..	5 × 2·5 × 1 mm. ..	c̄ correction 6/6 J. 1..	Traumatic cataract.
Outside sclerotic	8·5 × 5·5 × 3 mm. ..	6/6	F. b. traversed ciliary body vitreous and sclerotic, to reach its resting-place.
In lens ..	1·5 × 1 × ·5 mm. ..	c̄ correction 6/9 J. 1..	Small magnet failed.
Vitreous ..	.. ..	c̄ correction 6/9 ..	Traumatic cataract absorbed spontaneously.
Vitreous ..	6·5 × 2 × 1 mm. ..	c̄ correction 6/9 J. 1..	
Vitreous ..	4 × 1·25 × 5 mm. ..	c̄ astigmatic correction 6/6 J. 1.	
Vitreous ..	1·5 × 1 × ·5 mm. ..	c̄ correction 6/12 J. 1	Clear lens was wounded at operation.
Vitreous ..	3 × 2·5 × 2 mm. ..	6/12	Some vitreous lost.
Vitreous ..	.. ..	c̄ correction 6/12 ..	Traumatic cataract.
Entangled in iris	4·5 × 1·75 × 1 mm...	c̄ correction 6/18 ..	Vision deteriorated to 6/36. Malingerer. Lens unwounded.
Entangled in iris	2·5 × 1·5 × 0·5 mm.	c̄ correction 6/18 ..	There was optic neuritis at time of operation. Patient seen 3 months after operation.
Vitreous ..	4 × 1·25 × 0·5 mm...	c̄ correction 6/36 ..	Advergent eye, thick capsular membrane remains. Traumatic cataract.
Vitreous ..	4·5 × 2·25 × 1 mm...	6/36	Small magnet failed to withdraw f. b. Choroidal atrophy.
Vitreous ..	5·5 × 3 × 2 mm. ..	c̄ correction 6/60 ..	Patient is aphakic. There is a thick capsular membrane. Projection and field good.
Vitreous ..	2 × 1·5 × 1 mm ..	6/60	Opacities in lens present before operation increased afterwards.

No.	Patient.	Surgeon.	X-rays.	Operations.	Further operations.
18	Wm. K ..	Mr. Gunn ..	No	Haab's magnet. Small magnet. Iridectomy	.. ..
19	Charles C..	Mr. Morton ..	Yes	Division anterior synechia. Haab's magnet	.. ..
20	Fred. O. .	Mr. Morton ..	Yes	Haab's magnet. Small magnet. Iridectomy	.. ..
21	Wm. S. ..	Mr. Morton ..	Yes	Haab's magnet..	.. ..
22	Harry H. ..	Mr. Tay ..	No	Haab's magnet. Small magnet	.. ..
23	Wm. M. ..	Mr. Lawford	No	Haab's magnet. Curette evacuation	Curette evacuation
24	—	Mr. Lawford	No	Haab's magnet. Small magnet	.. ..
25	Wm. C. ..	Mr. Marshall	No	Haab's magnet. Iridectomy..	.. ..
26	Charles P...	Mr. Fisher ..	Yes	Haab's magnet. Small magnet	.. ..
27	Wm. W. ..	Mr. Marshall	No	Haab's magnet..	Iridectomy Curette evacuation
28	Wm. B. ..	Mr. Morton ..	No	Haab's magnet..	Division synechia Iridectomy
29	David A....	Mr. Collins ..	Yes	Haab's magnet. Iridectomy..	. ..
30	Frank P. ..	Mr. Spicer ..	No	Haab's magnet. Iridectomy..	Curette evacuation
31	Robt. S. ..	Mr. Marshall	No	Haab's magnet..	Needling. Curette evacuation
32	Herbert A.	Mr. Marshall	Yes	Haab's magnet..	Division synechia..
33	Wm. M. ..	Mr. Spicer ..	No	Haab's magnet. Small magnet	.. ..
34	Charles G.	Mr. Spicer ..	No	Haab's magnet..	.. ..

The removal of a f. b. from the a. c. or from the lens, without causing irretrievable damage to the eye, is an easy matter compared with the removal of a f. b. from the vitreous. In the majority of cases in which Haab's magnet was used,

Site of f. b.	Size of f. b.	Vision.	Remarks.
Vitreous ..	1·5 × 1 × ·5 mm. ..	Hand movements ..	Traumatic cataract to be dealt with later.
Vitreous ..	2·25 × 2 × 1 mm. ..	Hand movements ..	Small magnet failed. Traumatic cataract. Retina probably detached.
Vitreous ..	4·5 × 4 × 1·5 mm. ..	Counts fingers at 1 metre	Iritis and cyclitis at time of operation. Lens clear.
Vitreous ..	3 × 2 × 1·5 mm. ..	.. ..	Traumatic cataract.
Entangled in iris	.. ..	Hand movements ..	An old injured aphakic eye. When last seen some months ago patient had vesicular keratitis.
Vitreous ..	.. ..	Excision.	
Vitreous ..	.. ..	Excision.	
Vitreous ..	5 × 3·5 × 2 mm. ..	Excision	Iritis at time of operation.
Vitreous ..	3·5 × 2·25 × 1 mm. .	Excision	Shrunken globe, excised 2 months after operation.
Vitreous ..	7 × 1·5 × 1 mm. ..	Excision.	
Vitreous ..	6·5 × 4 × 1 mm. ..	Excision.	
Vitreous ..	2 × 1·5 × 1 mm. ..	Evisceration	The small magnet failed. Iritis at time of operation.
Vitreous ..	6 × 2·5 × 1 mm. ..	Excision.	
Vitreous ..	4·5 × 3 × 1·25 mm. .	Excision.	
Vitreous ..	20 × 5·5 × 3 mm. ..	Excision ..	f. b.'s of enormous size.
Vitreous ..	15 × 5	Excision ..	
Vitreous ..	3·5 × 3 × 1·5 mm. ..	Evisceration	Iritis.

the f. b. was in the vitreous, and these cases only will be considered further. They are 29 in number, and may be classified according to the visual acuity obtained after operation, as follows :—

(*a*) Those cases which attain to 6/12.

(*b*) Cases in which by further operation, such as extraction of an opaque lens, or needling of a capsular membrane in an aphakic eye, it may be expected that good vision may be attained.

(*c*) Cases in which vision is present, but in which so much damage has been done by the entrance of the f. b. that further improvement of vision cannot be hoped for.

(*d*) Cases in which after operation the eye was excised.

I.

Haab's Magnet Results. (*In Vitreous only.*)

	No. of cases.	Per-centage.
(*a*) Good vision	9	31
(*b*) Vision, with possibilities of improvement	5	17·2
(*c*) „ without „ „	3	10
(*d*) Excisions	12	41·3

From this table it is seen that in 58 per cent. of the cases in which Haab's magnet removed f. b.'s the eyes were saved, and that in half of these cases good vision was obtained.

In the Ophthalmic Hospital Reports, vol. xiv, Mackenzie has given the results obtained by using the small magnet for removing f. b.'s from the vitreous. Only 26 per cent. of the eyes were saved. Mackenzie's paper, however, was written before the days of X-ray localisation, whereas many of the Haab's magnet cases were X-rayed previous to the extraction of f. b.'s. Comparison, therefore, is not quite fair, since the prognosis in magnet extraction cases is more favourable if precise localisation has been carried out before the application of the magnet. It is then possible in many cases so to direct the magnetic force that a minimum of damage is done during the exit of the f. b. from the globe. The damage likely to be caused may be

(*a*) Entanglement of f. b. in ciliary body or iris.

(*b*) The injury of a hitherto unwounded lens,

(*c*) Prolapse of iris.

At Moorfields, the localisation of f. b.'s by the aid of X-rays has been carried out by Dr. Mackenzie Davidson, with great exactness.

A table is given below showing the results obtained in the 14 cases in which the f. b.'s had been localised exactly previous to their removal by Haab's magnet. Localisation was effected by X-rays in all the cases except one, and in this the f. b. could be seen with the ophthalmoscope.

II.

Haab's Magnet Results after X-rays. (In Vitreous only.)

	No. of cases.	Per-centage.
(*a*) Good vision	5	34·27
(*b*) Vision, with possibilities of improvement	4	25
(*c*) ,, without ,, ,,	2	12·5
(*d*) Excision	3	21·4

From comparison of Tables I and II it is seen that localisation has reduced the percentage of excisions by one-half, and has correspondingly improved the results in the eyes saved.

III.

Comparison of Small Magnet and Haab's Magnet Cases after Localisation. (In Vitreous only.)

	Small magnet.		Haab's magnet
	No. of cases.	Percentage.	Percentage.
(*a*) Good vision	3	16·6	34·27
(*b*) Vision, with possibilities of improvement	6	33·3	25
(*c*) Vision, without possibilities of improvement	5	27·7	12·5
(*d*) Excision	4	22·2	21·4

From Table III it is seen that the percentage of excisions as compared with the percentage of eyes saved is about the same with the two magnets, but that the quality

Small Magnet Cases after

No.	Patient.	Surgeon.	X-rays.	Operations.	Further operations.
1	Harry J. . . .	Mr. Lang.	Yes	Scleral incision. Small magnet	..
2	Roland L. . .	Mr. Collins . . .	Yes	Scleral incision. Small magnet	..
3	Arthur S. . .	Mr. Collins . . .	No	Small magnet	..
4	John M. . . .	Mr. Morton . .	Yes	Scleral incision. Small magnet failed	2nd small magnet trial successful
5	James D. . . .	Mr. Morton. . .	Yes	Small magnet	Curette evacuation
6	Alfred B. . .	Mr. Tay.	Yes	Small magnet	..
7	John A.	Mr. Tay.	Yes	Iridectomy. Small magnet. Curette evacuation	..
8	Thos. B. . . .	Mr. Morton . .	Yes	Small magnet.	Division anterior synechia
9	Henry R. . .	Mr. Collins. . .	Yes	Scleral incision. Small magnet	..
10	Robt. R. . .	Mr. Silcock . . .	Yes	Scleral incision. Small magnet	..
11	Henry P. . .	Mr. Tay.	Yes	Scleral incision. Small magnet	..
12	James T. . . .	Mr. Lawford. .	Yes	Scleral incision. Small magnet	..
13	Alfred M. . .	Mr. Tay.	Yes	Scleral incision. Small magnet	..
14	James F. . . .	Mr. Tay.	No	Scleral wound. Small magnet	..
15	Alec M. . . .	Mr. Lawford. .	Yes	Scleral incision. Small magnet failed	Excision. . . . .
16	Jacob K. . . .	Mr. Lang	Yes	Small magnet unsuccessful. . . .	Excision. . . . .
17	Fred. Y. . . .	Mr. Tay.	Yes	Scleral incision. Small magnet	Excision . . . . .
18	Wm. S.	Mr. Silcock . . .	Yes	Scleral incision. Small magnet	Excision. . . . .
				Case in which the Small Magnet failed	
	Alec M. . . .	Mr. Lawford .	Yes	Scleral incision. Small magnet failed	Excision. . . . .

Localisation. (In Vitreous only.)

Site of f. b.	Vision.	Remarks.
Vitreous	6/9 J 1	Seen 2 years after operation. Operation 14 days after injury.
Vitreous	6/9	Operation 10 days after injury.
Vitreous	6/9 J 1	Injury some days before operation. F. b. seen ophthalmoscopically. V. taken 3 years after operation.
Vitreous	6/36	Posterior polar opacity now present, 2 years after operation. Immediately after operation V. = 6/9. Operation day after injury.
Vitreous	$\bar{c}$ correction 5/60	External rectus paralysed, dense capsular membrane. Injury 4 weeks before operation.
Vitreous	Counts fingers at 1 metre	Traumatic cataract is present.
Vitreous	$\bar{c}$ correction 6/60	Injury 4 months before operation.
Vitreous	$\bar{c}$ correction < 6/60	Aphakia. Thick capsular membrane. Patient was seen 2 years after accident. Operation on day of injury.
Vitreous	P.L.	Seen 4 years after accident. Retina detached Injury 3 days before operation.
Vitreous	Hand movements.....	Injury 8 days before operation.
Vitreous	Counts fingers at 1 metre	Seen 2 years after operation. Lens has absorbed, thickened capsule alone remains.
In coats of eyeball	Hand movements.....	Haab's magnet tried before localisation of f b. After localisation, site of f. b. cut down upon and f. b. found in coats of globe. Injury 18 months before operation.
Vitreous	P. L.	Seen 3 years after accident. Projection bad. T. −. Injury 8 days before operation.
Retina...	Eye saved	F. b. seen ophthalmoscopically. Injury 3 days before operation.
Vitreous	..	F. b. found in globe.
Vitreous	..	Injury 12 days before magnet operation.
Vitreous	..	Accident 12 days before operation. Sympathetic ophthalmia resulted, causing vision in uninjured eye to fall from 6/6 to 6/24.
Vitreous	..	Seen first 14 days after accident.
to remove f. b. from Vitreous.		
Vitreous	..	F. b. found in excised globe.

of the results after using Haab's magnet is very much better than after using the small magnet.

It is interesting to compare the results obtained by the use of Haab's magnet after X-ray localisation with those obtained by the use of the small magnet after X-ray localisation.

The foregoing list of cases comprises all those treated at Moorfields, up to the end of July, 1901, in which f. b.'s have been removed by the small magnet after localisation by X-rays or the ophthalmoscope.

For the calculation of percentages the number of cases that I have been able to collect is really too small; but at any rate some idea may be obtained from them as to the respective value of the two magnets.

Until I compiled these tables I was of the opinion, from practical experience of the use of the two magnets, that the Haab's magnet results would entirely overwhelm those of the small magnet. This, however, is not the case. There are, nevertheless, several reasons why the Haab's magnet results do not appear so good from statistics as they really are in practice.

(1) In several of the Haab's magnet cases the eye had been so much damaged by the entrance of the f. b. that one could not hope to preserve it as a useful organ. In the small magnet cases the injury was, as a rule, slighter.

(2) As a general rule a longer period between the date of injury and the date of operation had elapsed in the small magnet cases than in the Haab's magnet cases.

(3) Statistics do not show one great value of Haab's magnet, which is, that if a f. b. is present in the vitreous it can almost surely be removed *viâ* the pupillary area by the magnet. This cannot always be done by the small magnet.

Method of using Haab's Magnet.

In using the Haab's magnet the patient is seated on a stool with the injured eye on the same level as the pole of

the magnet, and at a distance of about a yard from it. The current is then switched on at its minimum strength, and the patient is approached to the magnet until the injured globe is almost touching the magnet terminal. The current is then gradually increased to the maximum or until the f. b. gives evidence of its presence, either by appearing in the a. c. or by bulging forwards the iris at some point. If the f. b. has been localised it is possible to so direct its course by varying the position of the injured eye (thereby altering the relative position of magnet terminal, f. b., and structures of the globe), as to avoid injury during the exit of the f. b. While it is often necessary to use the full strength of the magnet in order to start the f. b. from its resting-place in the globe, it is generally best to diminish the current as soon as a certain momentum has been established, or as soon as the iris has bulged. This is especially the case where, owing to the lens being unwounded, it is impossible to direct the f. b. directly into the pupillary area. The reason for diminishing the current is that the less the momentum of the f. b. the less chance is there of the f. b. becoming entangled in the iris. If, in spite of these precautions, entanglement in the iris does occur, the current should be broken, and having directed the force of the magnet in such a way as to avoid the obstacle, it should be made again, using the minimum current.

I will now suppose that the f. b. has been drawn into the a. c.; the next step is to remove it thence. If there is a recent wound, the obvious thing to do is to remove it by that outlet.

If the wound has healed, or if the original wound of entrance was through the sclerotic, a corneal section must be made. This can be made either peripherally or nearer the centre of the cornea. When a peripheral section is made, the patient is placed on the operating table, and a keratome is used for the operation; the f. b. is removed from the a. c. by the small magnet. When the more central section is made, a Graefe's knife is used, the surgeon standing behind

the patient, who sits in a chair in front of Haab's magnet. The f. b. is then removed by the same magnet.

The peripheral incision is that more usually employed at Moorfields. The only inconvenience about it is that it necessitates the use of the small magnet, which is a somewhat clumsy instrument, and the use of which implies the multiplication of instruments. The advantage of the method is that the patient is already lying on the table prepared for any further operative procedure, such as iridectomy of prolapsed iris, &c.

In favour of the more central section it may be said that two instruments only are used in contact with the eye ; one is the terminal of the magnet, which has of course been boiled, and the other is the Graefe knife—speculum and fixation forceps being dispensed with. Again, the terminal of Haab's magnet need not usually be made to quite touch the eye.

In some cases it is impossible to attract the f. b. into the a. c. at all. The cause of this may be one of several conditions :—

(*a*) Entanglement of f. b. in iris or ciliary body.

(*b*) Entanglement of f. b. in coats of injured eye.

(*c*) The large size of the f. b.

In these cases a scleral incision must be made and the terminal of the magnet approximated to the gap produced. Haab's magnet should, at any rate, be tried first of all, for its terminal need not be introduced into the vitreous, whereas the terminal of the small magnet must be inserted between the lips of the wound, much disturbance of the vitreous being thereby produced.

In connection with the above operative procedures, it may be mentioned that subsequent operations for visual purposes on the eye in the cases of extraction of f. b.'s of Haab's magnet have been uniformly successful when they have been performed at a date some months after the primary operation.

The Royal London

Ophthalmic Hospital Reports.

Vol. XV. Part III. January, 1903.

I. ON THE DISTRIBUTION OF THE CHOROIDAL ARTERIES AS A FACTOR IN THE LOCALISATION OF CERTAIN FORMS OF CHOROIDITIS AND RETINITIS.*

By E. Nettleship.

Is there reason to think that the arrangement of the arteries in the choroid may affect the distribution of the changes in some forms of localised choroiditis and retinitis?

Two groups of cases are especially suitable for consideration in this respect, viz.:—(A) The whole family of central, usually senile, choroido-retinitis, not associated with any particular constitutional state; and (B) retinitis pigmentosa and cases of syphilitic retinitis accompanied by ring-scotoma.

In the family of central senile choroido-retinitis we can easily distinguish four well-marked clinical types though, as would be expected, mixed forms are met with. The types are (1) central guttate choroiditis, which is almost certainly the clinical equivalent of the so-called colloid nodules of the elastic lamina. This condition was first described by Mr. Hutchinson† in 1875, but has generally been called Tay's choroiditis at this hospital, from the conspicuous part ascribed

* The substance of this paper and the next formed the subjects of a Clinical Lecture given at the Moorfields Hospital on October 8th, 1902.

† R. L. O. H. Reports, vol. viii, p. 230, 1875. Goldzieher also wrote on similar cases before the publication of Fuchs's paper on Retinitis Circinata.

by Mr. Hutchinson in his paper to Mr. Waren Tay's observations and help. Some of the cases described in that paper showed other changes, but in nearly all of them the type of disease was that shown in Diagram 1,* taken from the Ophthalmological Society's Transactions, vol. iv, Pl. 2. In many cases the fovea itself is less affected than the perimacular region, a more or less complete belt of the spots encircling the disc and yellow-spot region, whilst the yellow-spot itself is comparatively free; but the yellow-spot is sometimes fully affected. Vision is damaged in various degrees, and since Tay's earlier observations were made many cases have been seen in which it was scarcely, if at all, lowered even when the actual macula was much involved: thus in the case from which Diagram I was taken V was 6/6 with each eye.

Type 2. An area of epithelial atrophy occupying the yellow-spot and more or less of the surrounding part. In well-marked cases the border is sharply defined, and shows a variety of irregularities, though the general shape is nearly circular or horizontally oval. (The diagram (No. 2) showing this form was taken from an original drawing.) There are varieties in which the shape is less regular, the border less defined, and the surface more pigmented. I do not know what morbid appearances precede the disturbance and removal of the pigment epithelium in this form of disease—perhaps hæmorrhage in some cases, superficial inflammation in others.

In Type 3 both the epithelium and choriocapillaris are destroyed, and the large vessels laid bare, these vessels themselves being usually much altered by opaque thickening of coats and contraction of lumen, and the blood-column in extreme cases either obliterated or hidden by the opacity of the coats. The diseased area is sharply defined, circular or oval in shape, and may be so large as to extend up to the disc. There is, in the typical cases, no collection of pigment.

* The references here, and further on, are to diagrams shown at the lecture.

(Diagram 3, showing this type, was taken from the Ophthalmological Society's Transactions, vol. iv, Pl. 8. There is also a good illustration in Jaeger's Large Atlas, second edition, Pl. 51.)

Type 4. In a fourth variety the corresponding part of the fundus is occupied by an oval or circular patch of densely opaque white, greyish-white, or yellowish-white substance lying beneath the retina and apparently upon the choroid. So far as I know, the origin and course of this form has not been worked out, nor is the structure of the patch known; the substance is, however, of appreciable thickness, as shown by ophthalmoscopic estimation and by the course of the retinal vessels. (The Diagram No. 4 of this form was from a drawing made by Mr. Holmes Spicer, and kindly lent by him.) Major Yarr* has lately published a drawing from a case that I think must have been an example of this deposit form of choroiditis, though he calls it choroidal atrophy; it shows, what is not uncommon in these cases, retinal vessels dipping into the mass and disappearing, doubtless anastomosing with vessels of the subjacent choroid. (Diagram 5 was copied from Yarr's plate.) The edge of the patch in Yarr's case is less regular than usual, but as, according to the history, it was of only about four months' duration, this is not surprising. Such a vascular communication† was shown also in two other diagrams taken from rough sketches of similar cases in my note-books (P. 12, 142, and P. 30, 37), and in one of these the patch of deposit was bordered by extravasated blood.

Another diagram was shown to illustrate the areolar form of atrophy (Type 3) emerging from a condition of previous deposit (Type 4), the deposit in the case from which this diagram was taken being in process of absorption instead of organising into dense tissue (P. 35, 52). That

* Yarr, Trans. Ophth. Soc., vol. xix, Pl. ix, Fig. 2.

† Retino-choroidal anastomoses occurring near the y. s. in cases of gross choroidal exudation were recorded by Loring in Knapp's Archives, vol. ii, 2, p. 1, in 1872.

such deposit may, on the other hand, be permanent is proved by a case published by Mr. Hartridge,* where a similar form of disease was watched for 14 years and seen to undergo little if any change.

In connection with these types of central choroiditis mention must be made of the retinitis circinata of Fuchs. It is ophthalmoscopically a disease of the deep retinal structures, and Ammann has found in microscopical sections blood between retina and choroid, and very marked disease of some of the neighbouring choroidal arteries; though he concludes that the hæmorrhages were derived from retinal vessels.†

My own experience is that, ophthalmoscopically, many mixed forms are found between typical retinitis circinata and one or other of the choroidal affections I have just alluded to. Even in Fuch's classical paper, his Fig. 7 (retinal) and Fig. 8 (choroidal) are from the two eyes of the same patient. I believe that even in typical cases of retinitis circinata the deeply-seated smoky-looking haze of the macular part of the retina is consistent with a disturbance of its choroidal nutrition, and that the later retinal appearances may be secondary.

I do not consider that the plate in Oeller's Atlas of Rare Ophthalmoscopic Conditions (C. Table I) with that of the earlier conditions of the same eye (C. Table VI) represent what Fuchs intends to convey by retinitis circinata, nor what most observers recognise as such, for the circinate arrangement here was preceded by obvious obliterative changes in the retinal arteries and coarse hæmorrhagic retinitis—conditions not seen, so far as I know, in what is usually recognised as the early stage of retinitis circinata.

The clinical course of the various types and their rela-

* Ophth. Soc.'s Trans., vol. xxi, p. 80, 1901.

† See Ammann, Knapp's Archives, vol. xxvii, p. 203, 1898 (from German edition, 1897). See also Goldzieher on his priority over Fuchs. Knapp's Archives, vol. xxviii, p. 638 (from German edition, vol. xxxiv, 1897).

tion to each other has not been fully investigated, and except Ammann's observations I know of no microscopical examinations. Observations of these various types of central change followed out from the commencement are much wanted but are difficult to get; the subjects seldom seek advice early, and often disappear when they find their sight getting no better; they are for the most part elderly, sometimes quite infirm; and again it often happens that the final details of the fundus changes as they proceed become more or less obscured by opacities in the lens. All the above forms of disease are, as implied in their nomenclature, usually confined to the centre of the fundus, but in all of them disease of the choroid is occasionally found elsewhere.

Has anyone watched the onset of Tay's guttate central choroiditis and seen it either increase or diminish? In one case where I found plenty of these spots in both eyes of a lady, aged 46, in 1886 (P. 12, 216), I believe I can say that the disease had spread over a somewhat wider area, and the individual spots become decidedly larger during the next 10 years; but I speak cautiously as to these points, because in the meantime the patient's lenses had become somewhat cloudy.

In regard both to Tay's disease and the three areolar types examination will, as already stated, lead to the frequent observation of intermediate and mixed forms and to the alliance of some of them with retinitis circinata; at least I feel sure we must not yet assume that the central areolar atrophy with thickening and narrowing of choroidal vessels (Type 3) is always atrophic from the beginning.* Some cases both of that and of the epithelial type may be simply atrophic, but it is more likely that in most of them a stage of exudation or inflammation precedes. Indeed, it is easy to understand that a similar disease of vessel-walls may,

* Ammann, in his paper on Retinitis Circinata, thinks the perivascular lymph-spaces may be primarily to blame in that disease. He is of course not speaking of the areolar choroidal atrophy, but the speculation may be extended to that form.

according to its degree or distribution, lead to mere atrophy in one case, but to œdema, hæmorrhage, and other changes, followed by atrophy or by organisation into new tissue, in another.

Why should the changes in this family group be so constantly limited to the area that includes the yellow-spot and optic disc? Probably the blood-supply has something to do with it, the blood-supply itself being related to the high structural and functional perfection of the retina at this part of the eye.

The capillary network both of the choroid and retina is much closer at the yellow-spot than in any other part. Whether the rapidity of the blood-stream through the close capillary mesh-work of the central area is as great as where the meshes are coarser, depends presumably on the dimensions and number of the respective arteries. In regard to the retina, Mr. Parsons* tells us that, relatively to the capillaries, the arterial supply for the central area is less than for other parts; if this be so a slight thickening of the coats of these arterioles would have serious consequences to the inner retinal layers. The corresponding part of the choroid, by which the outer layers of the retina are nourished, is well supplied by the short posterior ciliary arteries, though the great variability in their number as they penetrate the sclerotic may lead to variations in the effectiveness of the supply in different eyes. However this may be, we can hardly doubt that contraction of the lumen of one or more of the short ciliaries by endarteritis or other chronic change would soon tell on the integrity of the corresponding choroidal area; whilst if the chief inner or outer ciliary branch of the ophthalmic artery, from which the short ciliaries arise, were diseased, the area fed by all of its branches would of course be affected.

I suspect, therefore, that central senile choroido-retinal disease in its various manifestations is grounded chiefly upon disease of the posterior ciliary arteries—either the

* Parsons, Ophthalmic Review, vol. xx, p. 188. (July, 1901.)

trunk vessels or the branches that perforate the sclerotic near the optic nerve,—and that any affection of the retina itself is usually consequential If this be so, we can readily understand why the disease is confined to the central area of the fundus. The process being non-infective, and conditioned by blood-vessels, differs radically from those kinds of choroiditis that are due to blood-state. In most cases of inflammation of the uveal coat and of the cornea due to a morbid blood-state the process tends to spread forward or backward to other parts, the amount of such extension being largely a question of the length of time that the malady remains active.

In so-called retinitis pigmentosa and in some cases of syphilitic retinitis the distribution of the disease is, I think, dependent in part on the arterial system of the choroid at the equatorial part of the globe.

In retinitis pigmentosa the ophthalmoscopic changes are often most conspicuous at, and sometimes confined to, the equatorial belt, ceasing not only behind it but in front also. In these cases the field shows a belt or portions of a belt of blindness between the seeing centre and the seeing periphery. In syphilitic retinitis, too, such a ring-scotoma or blind belt can not infrequently be found if sought for. (Diagrams were shown.)

It is impossible to explain this feature of these two diseases by anything in the structure of the choroid or retina. But it might be explicable if we could show that the supply of blood to the choroid was less efficient at the equator than either at the posterior or anterior parts of the fundus; and I think that this is the case. The posterior part of the choroid is supplied, and well supplied, by the short posterior ciliary arteries; the anterior part is supplied to a great extent by recurrent branches from the long posterior ciliary and anterior ciliary arteries. The terminal twigs of these anterior and posterior systems meet, and to a certain extent anastomose, at the equatorial region. The equator seems to

be a sort of "divide" between the two systems, and to be not very efficiently served by either, whilst the current must be slowed or reversed in many of the anastomosing twigs.

Given a congenital tendency (as in retinitis pigmentosa) to atrophy or hardening of the inner layers of the choroid, it would probably tell first where the nutrient stream was weakest; whilst if, owing to minor variations in the arterial arrangements just mentioned, some parts of the equator were better supplied than others, the incompleteness of the blind belt in certain cases would be explained. The explanation here suggested for the blind belt in the field in retinitis pigmentosa assumes that that disease takes its origin in the choroid, and this agrees I believe with the most recent views.

In order to explain the ring-scotoma of syphilitic retinitis by vascular supply, we must assume arteritis of the equatorial twigs of the choroidal arteries, either of the posterior or anterior system, or both; not on the whole a too strained assumption. Syphilitic retinitis is so very often accompanied by visible choroiditis, that even when at first no choroiditis can be proved by ophthalmoscopic examination, we may often safely prophecy that it will become visible later when the retina has cleared or become atrophic.

II. ON THE INFLUENCE OF OVER-USE AND FATIGUE OF THE EYES IN CAUSING ORGANIC DISEASE OF THE RETINA AND CHOROID.

(After allusion to fatigue of the eye-muscles from reading, writing and sewing, from fixed gazing, especially gazing upwards, and from rapid movements in following the objects passed in travelling; to the effects of evaporation, exposure and the friction of the eyelids, in producing troublesome mixed forms of asthenopia; and to iritis as often kept up or prevented from recovering, but seldom actually brought on, by overuse of the eyes; attention was particularly directed to the choroid and retina).

In regard to the choroid and retina the only case that, with our present knowledge, can be with certainty attributed to overuse is the "blinding" by Direct Sun-light, in which a small central scotoma follows immediately, or within a few hours, after looking at the sun without any, or with insufficient, protection. Good, though not quite perfect, recovery of vision usually follows after weeks or months, unless the primary lowering of visual acuity was very marked, but V. $\frac{1}{3}$ or less is seldom followed by satisfactory recovery.* The ophthalmoscope reveals either no visible changes or slight alterations of colour and clearness at the yellow spot, such as might be caused by œdema of retina and choroid and alteration in the quantity and distribution of the pigment granules of the retinal epithelium. In fact, experiments on the eyes of rabbits and frogs made under much severer exposure than can ever occur in the human eye, have shown that destruction of the rods and cones, exudation between retina and choroid and choroidal hæmorrhages are amongst the immediate results, whilst pigmented scar tissue may be a later product.†

* Mackay, Ophthalmic Review, vol. xiii, 1894, p. 92.

† Czerny and Deutschmann, according to Mackay, *loc. cit.*, p. 83.

The same cause, gazing at the sun, was believed to produce thrombosis of a branch of the retinal artery and other retinal and even choroidal changes, in a case published by Mr. Batten.*

Dr. Oliver, in 1897,† published a case in which choroido-retinal changes followed in both eyes after exposure to an extremely brilliant flash of lightning. In this case the R. eye was affected immediately or within a few hours, and showed marked fundus change nine days later; but the vision of the L. was unaffected till 10 days after the exposure, whilst fresh retinal hæmorrhage occurred in it nearly three months later. Though this young man's urine and heart were examined and found normal, I do not find any mention of syphilis in the record, and this omission much diminishes its value in the present connexion.

A case in which one eye became nearly blind with partial atrophy of the optic nerve, apparently from primary damage to the macular part of the retina, the other eye suffering for a time, but recovering with slight changes at the macula, was published by Mr. Treacher Collins‡ in 1896. The symptoms (central scotoma in each eye) in this case followed immediately after the patient (a woman aged 49) had stared at the sun, without protection, for five or ten minutes. It is noteworthy in this case that the failure of vision did not reach its worst till some days after the exposure, and also that the eye in which permanent, presumably ascending, damage occurred to the optic nerve, showed less visible change at the yellow spot than did the eye that recovered with a normal disc.§

A similar discrepancy between visual acuteness and ophthalmoscopic change in the two eyes was found in the case of a

* Rayner D. Batten, Trans. Ophth. Soc., vol. xxi, 1901, p. 70.

† Oliver, Trans. American Ophth. Soc., vol. vii, 1897, p. 613.

‡ Treacher Collins, R.L.O.H. Reports, vol xiv, 1896, p. 374.

§ Compare O. Haab on Secondary Atrophy of the Optic Nerves after Macular Disease, Abstract in Ophthalmic Review, vol. xxi, Oct. 1902, p. 287.

Japanese gentleman whom I saw in 1898 (P. 48, 57). He was then 28, and told me that 10 years earlier he had damaged his eyes by looking for an hour at an eclipse of the sun without any protection. On the same day, almost immediately after, he found a "black patch" in front of whatever he looked at, but could see to each side of it. The next day his R. eye was covered up, and kept covered for several weeks, and it got perfectly well. The L., which remained open and in use, and entirely untreated until the R. was cured, never entirely recovered. When I saw him, 10 years after the accident, V. of the R. was 6/5 with several ill-defined semi-confluent choroidal dots at or just below the fovea: V. of L. was only 6/9, but I could see no changes at the y. s. or elsewhere; the defect was not optical.

It seems, therefore, as Mr. Rayner Batten has remarked in his paper already referred to, that the amount of damage to the retina varies much in these cases, and is not to be measured only by the length of time that the exposure lasts. We may add that the damage apparently does not depend entirely upon the absolute brightness of the sun-light or its spectral composition, for the two eyes often suffer in a different degree from an apparently equal exposure.

Such idiosyncrasy of the individual, or lower resisting power in one eye than in the other, is especially noteworthy, because we find cases that, as regards symptoms and ophthalmoscopic changes, are indistinguishable from the typical sun-blinding cases, but in which only moderate over-illumination, or none at all, can be alleged. It is, I think, most likely that fatigue of the central part of the retina either by relatively too intense light, or by too prolonged use in ordinary light combined with very accurate focussing, furnishes the true explanation in many cases of this group, the patient being perhaps rendered specially susceptible by general overwork or debility.

Some years ago I recorded* the case of a busy physician, aged 50, who noticed sudden failure of central vision with

* Ophthalmic Review, vol. vii, 1888, p. 33; Case III.

positive scotoma in his L. eye a few hours after using that eye at a microscope illuminated by direct sunlight. The eye very nearly, but never quite, recovered. There was an area of disturbance of the pigment epithelium at the y.s. In the same paper the case* of a naval officer is mentioned, in whose R. eye a permanent defect with metamorphopsia came on one day after this eye had been used for three hours continuously at the telescope of his theodolite in marine survey work. Two years later I found V. 6/9 barely, with a number of fine dots of choroidal or deep retinal disease at the y.s. The other eye, which he had never used for the theodolite work was perfect in every way. About the same time Dr. Brailey† recorded a case of a gentleman who had only one working eye, and it failed rapidly after he had been engaged closely at fine pen and ink drawing; fine central choroido-retinal changes were found.

In another case, perhaps less conclusive (P. 30, 19), a medical man, æt. 59, with two good eyes, each with about 2·5 D of My., undertook for a wager to count how many times the letter "e" occurred in a pamphlet of twelve pages of print, the type about equal to 5 Jæger. It was in the winter, was to be done against time, and he did it mostly by artificial light in the evening. Either during or immediately after this competition, he found his sight wrong, and discovered that the L. was defective. It got gradually worse, and remained so. When I saw him four years later, V. of this eye, corrected, was only 6/60; the central retina was occupied by a nearly complete zone of small bright white deposits with a few small dark blood dots at the y. s.; the retinal vessels, which showed no visible alteration, were in front of the deposits; a sort of retinitis circinata in fact. The other (R.) eye, with correction, saw 6/9, and showed no disease. The urine had been normal throughout the case.

I have notes of a considerable number of cases like those above mentioned, where a small positive scotoma, with or without noticeable metamorphopsia and fine changes at the y. s.,

* Case 1.

† Brailey, Trans. Ophth. Soc., vol. vii, p. 177.

came on suddenly or quickly, but in which it could not be stated from the history that the eye or eyes had been either unduly worked or exposed to strong light. But in many such apparently causeless examples of acute central retino-choroiditis there may, as already suggested, have been relative overwork, or what is the same thing, lowered resisting power, owing to some condition of health or occupation.

It seems to me, therefore, that leaving the direct sunlight cases apart, we shall be wise to believe that prolonged use of the central part of the retina, especially for minute stationary objects, under strong illumination and with accurate focussing, can, in certain circumstances, cause organic disease of the retina or choroid or both, in the eye so used.

I believe also that fatigue or exhaustion of the retina and choroid sometimes causes, or at least contributes to, more severe and extensive disease than the form just described. In Case 5, in the paper just mentioned,* severe disseminated choroiditis, mostly central, came on immediately after exposure to glare, heat and wet in a young naval officer who was engaged all one night in combating a fire that broke out in his ship. His sight was much damaged. In Case 6, of the same paper, peripheral choroiditis with iritis and opacities in the vitreous occurred in a lad of 16 who had been doing heavy work out of doors, chiefly wood-sawing, in very hot, sunny weather in July; till then he had been at school; syphilis very improbable. In the next case,

(P. 18, 164).—A young lady of 19 or 20 suffered sudden failure of her L. eye after using a telescope, and soon after an attack of scarlet fever. She was told by an eminent ophthalmic surgeon that "effusion" was present; sight improved, but a cloud remained near the centre. At the age of 36, when pregnant, she was exhausted by having to nurse her father both day and night, and a miscarriage followed; the R. eye now failed rapidly, in one night, and she was told there was disease at the

* Ophthalmic Review, *loc. cit.*

back; the eye did not recover. Eleven years later, when 47, the L. (or first affected and now the only working eye) failed again; she had been sewing hard for hours one evening to finish some work for a bazaar, and found the dimness the next morning. I saw her a fortnight later (June 20, 1889), when 47 years old. In the L., V. was 6/60, and there were old choroidal changes and an obviously new focus of choroiditis, all in the central area. In the R. the changes were much more gross and extensive, and all old, and there were some iritic adhesions. She married at 24, and had nine children, all of whom lived, and one miscarriage.

(P. 19, 54).—An old lady went through much distress and strain at the age of 65, when her husband died, and at the same time her eyes broke down rapidly, so that after a few weeks she could neither read nor sew; no improvement took place. I saw her about nine months later, with very extensive superficial choroidal atrophy, chiefly central, but partly peripheral, and marked haze of the central retina. V. was only fingers at 1 to 3 metres.

A gentleman of 68 (P. 19, 24), suffering from general nervous exhaustion, underwent, in addition, a severe shock, and his sight failed rapidly in both eyes; he was told there was blood at the back. Ten months later I found much retinal hæmorrhage over the central area in both eyes, with some white patches in one. There was neither sugar nor albumen.

A lady, æt. 52 (P. 19, 192), with the cares of an establishment, added to permanent harass from the chronic nervous illness of her husband, went for a long drive one Saturday afternoon without a sun-shade, the sun shining directly on her face. That same evening she noticed a blur in reading, and found it was from the R. eye. I saw her three days later, March 28, 1893, and found a hæmorrhage and some white exudation in the retina up and out from the O.D., and just below the superior macular vessels. A fortnight later, the blood had gone, and the white patch was more sharply defined; later this quite disappeared, leaving the vein indented by an obviously thickened artery. Three years later (April, 1896), when she had one day been standing out in the sun directing some gardening operations, a similar attack occurred in the L. eye, and I found 6 days later a hæmorrhage and white effusion close to the lower-

outer edge of the O. D.; a month after this, almost complete absorption of blood and white effusion had taken place, and a vein at the centre of the blood area was seen to be crossed by an artery. Her physician considered her systemic arteries atheromatous and heart dilated, but always found the urine free from albumen. She was at one time liable to small extravasations, preceded by pricking pain, in the skin of the R. lower lid, and when I last saw her in 1899, she had had severe epistaxis. Her eyes were hypermetropic 4·5 D., but she began efficient glasses between 30 and 40. Once, when 30, she had gout in the foot, and had to give up beer.

In this case, exposure of the eyes to direct sun-light and heat produced on two occasions, we may suppose, general retinal congestion and rupture of a vein where it was crossed, and compressed, by a hard artery.

There is, I am sure, no doubt that in elderly myopic persons hæmorrhage, sometimes into the choroid, in other cases into the vitreous, is not uncommon as a result of combined overuse of the eyes and fatigue: the weakened vessel-walls of the myopic and senile choroid cannot bear the distension caused by excessive work or want of sleep. No doubt there may be contributory causes of congestion such as irregular eating and drinking, and carelessness as to the bowels, all of which are apt to occur at times of stress and exhaustion of whatever kind.

A spinster lady of 54 (P. 50, 106), much engaged in parish work, had had a specially hard winter with nursing and other work, and just before Easter she undertook a quantity of church and charity duty. On the Wednesday of Easter week her R. eye (with 14 D. of My.) failed, with black smuts. Two months later I found large webs in the vitreous and (old) choroidal changes at the y. s., besides a large annular staphyloma and marginal opacity in the lens; V. 6/36 with −14 D. The L., with My. 9 D. and V. 6/9, showed no changes anywhere except a narrow crescent.

A married lady, æt. 56 (P. 52, 85), was overwrought about the New Year, 1900, by the combined effects of influenza in her

house, a new cook who turned out a drunkard, and the death of an aunt. On January 12, 1900, she noticed a black thing before her R. eye, and on 19th (one week) I found large webs in the vitreous, but no other disease except a very moderate crescent V. with -8 D. and a cylinder 6/18. The L., with 2 D. less of M., but similar As., had 6/9, and all normal. Such cases might easily be multiplied.

Several of my cases of choroidal and vitreous disease, associated with overuse, and others of the same kind without proved overuse, have been in plethoric, excitable, very active women with high myopia, and aged between 40 and 50. None were known to be alcoholic, and I am sure that most of them were not so, but now and then in such a case the suspicion has arisen. Similar cases are seen in men, but less commonly. The relative frequency in women is probably related to the menopause.

There seems no doubt that detachment of the retina may occur in similar circumstances, even in normally formed eyes, and we can scarcely doubt the causal influence of the excessive functioning.

A bank clerk, æt. 28 (P. 54, 26), had his sixth attack of influenza—a bad attack—at the end of January, 1900. The influenza pulled him down very much, but he had to return to duty before he was strong, and in addition to make up arrears of work. Soon after this he noticed something wrong with his sight, and discovered it to be in the R. eye, where detachment of the retina was found shortly afterwards. His eyes were emmetropic.

An actuary, æt. 57 (P. 55, 231), with My. 7 D. in the R. and 6 D. in the L., got detachment of the R. retina in April, 1901, after working hard at a statistical paper and correcting the proofs of it: he had not been accustomed to work requiring such a strain upon his eyes, and seems to have felt it a good deal. The eye had doubtless been a weak one, for Mr. Lawford had found a web in the vitreous when the patient had a troublesome cough some three years earlier, but this cleared up long before the detachment occurred.

In many of the examples just cited, the eyes were no doubt predisposed to degenerative lesions, as appears not only from the details of some of the cases themselves, but from the well-known occurrence of similar mishaps in the absence of marked fatigue or overuse. But in the particular instances quoted, there seems little doubt that the mischief would not have happened when it did, and would perhaps not have been so severe, had there been no fatigue or exhaustion.

And here I may say that I have gradually come to attribute greater influence to fatigue and overwork, local and general, than to more directly mechanical agencies in the production of intraocular hæmorrhage and even detachment of the retina. I, of course, do not say that stooping, straining, coughing, vomiting and other causes of temporary hindrance to the return of venous blood from the head; or muscular exertion causing violent action of the heart; or blows on the eye or concussion either of the head or the whole body, take no share in causing such maladies, but I think their influence has been over-rated, at any rate in the past, and that too little attention has been bestowed on physiological congestion from overuse.

ON THE CHILDREN OF PATIENTS WHO HAVE HAD INTERSTITIAL KERATITIS.

By E. Treacher Collins.

The difficulty of obtaining absolute proof of the possibility of transmission of syphilis to the third generation was well demonstrated in an interesting paper by Dr. George Ogilvie, published in The British Journal of Dermatology in 1897. In it all the recorded evidence on the subject up to that date was critically examined. Dr. Ogilvie divided the subject into first, the possibility of the transmission of actual syphilis, and secondly, the production of a lowered state of vitality, "a dystrophic influence," "parasyphilis."

As regards the first, after having pointed out the difficulty of obtaining absolute proof on many points in connection with syphilis, upon which nearly all competent observers are agreed, he says, "In the same way we have to look upon the question of syphilitic transmission to the third generation. We have quoted above expressions of the two extreme opinions held on this subject. On the one hand we are told that 'there is not the slightest evidence' of such transmission; on the other, that 'there are undoubtedly cases' in which syphilis was transmitted. Both statements are equally unwarranted. The evidence before us furnishes us, if not with absolute proof, still with reasonable probability, that syphilis may descend to the third generation. On the other hand, there is no evidence to support the possibility of transmission to the fourth generation."

As to the second point, he sums up as follows:—"Without denying that the 'dystrophic' influence of syphilis may make itself felt in some way or other in the third generation, I consider that up to now statements to this effect are more or less vague conjectures, and incapable of being proved by clinical evidence. Nor do I see how we shall emerge

from this state of uncertainty, without that extensive co-operation of the general practitioner, which seems as unattainable as it would be of supreme value."

On these very important but undecided matters, it occurred to the writer that some data might be obtained, by collecting notes of the family histories of women, presenting themselves at an Ophthalmic Hospital suffering from interstitial keratitis, and having at least one other sign of congenital syphilis, who were married and had children.

There could be little doubt that these children would be the descendants of a grand-parent who had had primary syphilis; their number and condition of health could be ascertained, and some of them could be had up for examination.

To determine definitely whether or not the father of these children had had primary syphilis, would have been a matter of considerable difficulty, and it was decided not to attempt it.

So that the presence of syphilitic symptoms in the children themselves would not be proof of transmission to the third generation. The occurrence, however, of such symptoms in a number of the children from different families would be a very suggestive piece of evidence. On the other hand, the absence of syphilitic symptoms from the large majority of children from different families would definitely prove that women, the subjects of inherited syphilis, may have children perfectly free from any actual syphilitic taint.

The number of the woman's brothers and sisters living and dead could be noted, and a comparison could then be made as to the amount of mortality in the children of the two generations.

A comparison could also be effected as to the mortality of infants in the third generation, and the average mortality for children under one year of age given in the Registrar General's returns for the whole of London for some years past. This would afford some evidence as to the presence or absence of "dystrophic influence."

As the second decade in life is the one during which interstitial keratitis most frequently occurs, it necessarily took some time to collect a sufficient number of cases in which it appeared so late in life as to have allowed of the patients becoming mothers.

It was thought well to wait until twelve had been obtained, to render the data of sufficient value for publication.

The following twelve cases, the details of which have been arranged in tabular form, have all come under the writer's care as out patients at the Royal London Ophthalmic Hospital during the past six years.

Unfortunately, in the rush of out-patient work, in one or two cases some points have been omitted from the histories, and the patients have since passed away from observation.

The youngest of the twelve was 20 years of age, and the oldest was 52 years. It might naturally be questioned whether interstitial keratitis occurring as late as 52 could really be hereditary syphilitic in origin, and the writer would have hesitated in including this case, had not the eye affection been of the usual type, and the patient presented the most typically notched teeth.

In none of the cases has the presence of interstitial keratitis been relied upon alone as a diagnostic sign of inherited syphilis, in all except one there has been besides either, scars at the angles of the mouth, notched teeth, or a suggestive physionomy. In the one case in which none of these other symptoms were noted, the fact that the patient was one of a family of 15, 12 of whom died in infancy, has been taken as sufficient confirmatory evidence of the presence of inherited syphilis.

The 12 women have had 60 children and five miscarriages between them. Thirty-four of the children are living, and 26 have died. Of the 34 living, 25, of ages varying from 19 years to 4 months, are stated to be healthy and have had no symptoms of a syphilitic nature. Fourteen of these have been examined by the writer, and appeared in excellent health and free from all deformities.

Of the remaining nine which may be termed unhealthy—

One has had what was called "congestion of the brain";

Five of one family are said to be frequently ill;

One has hydrocephalus;

One has had a rash on the skin, snuffles, ulceration about the anus, and lacrymal obstruction;

One of another family had snuffles in infancy.

Of the 26 that have died, 4 were still born; one died of measles aged 5 years, one of scarlet fever, age not stated, the remainder in infancy of such affections as "meningitis," "diarrhœa," "fits," and one of small pox.

From the foregoing analysis, it would seem fair to assume that as 25 out of the 34 living children are presumably healthy, and 14 certainly so, women the subjects of inherited syphilis may certainly have healthy children free from all taint of that disease.

One of the living children seems to have presented definite syphilitic symptoms, and three of the others symptoms which may have been syphilitic in origin. Without any history of the presence or absence of primary syphilis in the father, this cannot be taken as proof of the transmission of syphilis to the third generation. On the other hand, the small proportion of the living children who have had symptoms of a syphilitic character, is presumptive evidence against transmission to the third generation.

It would be obviously impossible to get any accurate statement as to the number of miscarriages the patients' mothers have had. So that no comparison of any value can be made between the number of the patients' and their mothers' miscarriages.

It is worthy, however, of note that among these 12 women who were the subjects of inherited syphilis, though they had 60 children, a history of only five miscarriages could be elicited.

Coming next to compare the mortality amongst the children of the two generations:—

It is found that out of 91 children of the mothers' generation, 44 were stillborn, or died in early childhood, 48·3 per cent.

Out of 60 children in the next generation, 23 were still born, or died in early childhood, 38·2 per cent.

The Registrar General gives the average proportion of deaths in London under one year of age, for the ten years from 1890 to 1900, as 160 per 1,000, 16 per cent.

In the histories of several of the cases here recorded, the actual age at which the children died is not reported, it being often merely stated that they died in infancy.

It is found that in the mothers' generation, out of 91 children, 36 died under one year of age, or are said to have died in infancy, 39·4 per cent.

In the succeeding generation, out of 60 children, 22 died under one year of age, or are said to have died in infancy, 36·6 per cent.

From the above figures, it would seem fair to draw the following conclusions:—

That the mortality amongst the children of parents who have had primary syphilis is somewhat greater than amongst the grand children.

That, after some allowance is made for the probability of some of those stated to have died in infancy having been more than one year old, the difference between the average infant mortality in London (16 per cent.), and that amongst the grand children of those who had primary syphilis (36·6 per cent.), remains very marked, so marked as to make it impossible to avoid the inference of the presence of some "dystrophic influence."

The smallness of the number of miscarriages in the 12 women the subjects of inherited syphilis would seem to show that the dystrophic influence does not make itself felt upon the fœtus *in utero*.

I. No.	II. Name.	III. Age.	IV. Evidence of inherited syphilis.	V. Family history of patient.	VI. Children of patient.
1	Annie C. .. 26/8/97	27	R. E. interstitial keratitis. L. E. nebulæ of cornea. Scars at angles of mouth; upper lateral incisors and canines absent, other teeth good.	The 3rd of a family of 5, of whom 3 have died, 2 of "consumption," æt. 21 to 22 years; one died in infancy.	Two children, one dead, no miscarriages. 1st child, boy, æt. 3½ years; seen, well developed and apparently healthy. Has had "congestion of brain" (? meningitis), was unconscious for about two days. Never any snuffles, said to have "breaking out on face at times." 2nd child, boy, died when 1¾ years old from "meningitis."
2	Rebecca M.. 29/10/96	52	R. and L. eyes interstitial keratitis. R. inflamed 3 months; L. 3 weeks. Typically notched teeth.	One of three children, one brother living, a sister died at the age of 24 years.	Twelve children, 7 of whom have died in infancy. The five living are said to be frequently ill, nothing very definite concerning their illness known, none of them seen.
3	Clara G. .. 10/1/98	26	R. E. divergent and sight defective many years, a few white spots at macula V. = 6/24. L. E. interstitial keratitis. Teeth notched, of screw-driver shape. Later R. E. also became affected with interstitial keratitis.	One of a family of 11, eight of whom died in infancy, and three of whom are living.	Four children, one dead, no miscarriages. 1st child, æt. 7 years; seen, a healthy-looking child, no deformities, teeth good; never any illness; never any rash on skin or snuffles. 2nd child died at age of 1¾ years of "diarrhœa." 3rd child, æt. 3 years; seen, a healthy-looking child, no deformities; never any illness; never any rash on skin or snuffles. 4th child, æt. 10 months, also seen, healthy, and no history of any illness.

I. No.	II. Name.	III. Age.	IV. Evidence of inherited syphilis.	V. Family history of patient.	VI. Children of patient.
4	Mary G. .. 5/12/98	38	R. E. interstitial keratitis.	One of a family of 15, twelve of whom died in infancy. Two sisters and one brother living, patient the youngest.	Fourteen children, 11 dead, 2 miscarriages. One died, æt. 5 years, of measles, two were stillborn, eight were born alive but died in infancy, some from "fits." The three living are æt. 19, 18, and 4 years respectively. The one æt. 4 seen, apparently quite healthy and well formed; the other two stated to have good health.
5	Mrs. F. .. 11/5/99	23	L. E. shrunken 14 years. R. E. interstitial keratitis. Physionomy and central incisor teeth suggestive of inherited syphilis.	One of a family of 4, one sister living; one brother and one sister died in infancy.	One child, no miscarriages. Child æt. 3 years, seen, healthy-looking and well developed, has never had any rash on skin or snuffles.
6	Laura J. .. 26/6/99	27	Interstitial keratitis both eyes. L. first inflamed 2 years ago. R. four months ago. Physionomy and teeth typical of hereditary syphilis.	.. .. ..	Four children, 2 dead (stillborn). Eldest living, æt. 4 years, seen; head hydrocephalic, never any fits; the younger æt. $1\frac{7}{12}$ years.

7	Emily D. .. 17/7/99	33	Interstitial keratitis both eyes. Scars at angles of mouth. Nothing typical about teeth or physionomy.	One of a family of 15, seven of whom are living; the others mostly died in infancy.	Five children, all living; "all healthy," no history of snuffles or rash on skin in infancy in any of them. The 2nd, æt. 10 years, seen; also the youngest, both in good health, no deformities, teeth good. The youngest has phlyctenular ophthalmia.
8	Mrs. P. .. 15/3/00	25	Signs of past interstitial keratitis in both eyes. Inflammation in them first four years ago, and again one year ago. Scars at angles of mouth. No deafness. Teeth not notched.	The 7th of a family of 10, six of whom are living. Her mother had 4 miscarriages before patient was born. The first four children are said to have died of smallpox, pneumonia, or violence. The 8th child is nearly blind with one eye.	Four children, 1 dead. 1st child died when 3 weeks old of smallpox. 2nd child, boy, æt. 5 years, had snuffles from birth till the age of 6 months; had a rash on the face and arms at age of 3 weeks, lasting about a week; pneumonia at the age of 2 years, and also ulceration about arms. Child seen; has lacrymal obstruction on the left side, eye said always to have been watery. No probe could be passed, bony obstruction, otherwise the child appears healthy. 3rd child, boy, æt. 4 years, seen; fairly healthy, no deformities, never any snuffles, rash on skin or sores on body. 4th child, æt. 4 months, seen; fairly healthy, never any snuffles or rash on skin.
9	Eliz. W. .. 31/5/00	20	L. E. interstitial keratitis. Physionomy suggestive of inherited syphilis. Scars at angles of mouth. Teeth not notched.	The eldest living of a family of 8. Three died in infancy.	One child, æt. 11 months, seen; apparently in excellent health, no deformities, never any rash on skin or snuffles.

I. No.	II. Name.	III. Age.	IV. Evidence of inherited syphilis.	V. Family history of patient.	VI. Children of patient.
10	Jane D. .. 9/7/00	39	L. E. interstitial keratitis. Scars at angles of mouth.	One of a family of about 7. Two brothers and one sister living. Three or more died in infancy.	Eight children ; 2 dead ; 3 miscarriages. One died of scarlet fever ; the other, " born blind," died, æt. 8 months. Eldest living is 19 years of age. One, æt. 3 years, seen ; in good health, well developed. None of the children have had snuffles or rash on the skin at birth.
11	Milly R. .. 25/4/01	26	Interstitial keratitis in both eyes. R. E. affected three years ago. L. E. 18 months ago. Scars at angles of mouth. Teeth not notched.	One of a family of 10. Three died in infancy, seven are living.	Three children, one born at 6 months, and died on second day. Eldest living is æt. 4 years, said to have had snuffles when a baby, no rash on skin at any time. Youngest, æt. 15 months. Both children seen ; strong, well formed, and healthy-looking.
12	Lilly A. .. 29/9/02	26	R. E. inflamed 5 years ago, nebulæ of cornea. L. E. affected 5 weeks, interstitial keratitis. Scars at angles of mouth. Upper central incisors wedge-shaped, and one notched, lateral incisors absent. Physionomy suggestive of inherited syphilis. No deafness.	The second of a family of three, all living.	Two children, both living ; no miscarriages. Eldest child, æt. 3 years, has had no illness except measles. Youngest, æt. 14 months, never any illness. Both children seen in good health, and no deformities.

THE HEALING OF WOUNDS OF THE RETINA, CHOROID, AND SCLERA, WITH SOME REMARKS ON THE PATHOLOGY OF "RETINITIS PROLIFERANS."

By J. Herbert Parsons, *Curator.*

The material upon which this research is based consists of experimental lesions produced in six monkeys, and three cases of injury by foreign bodies, in each case a piece of steel, which perforated the globe and led to excision. The latter are selected from a large number of such excised eyes which have been examined in the Pathological Laboratory of the Royal London Ophthalmic Hospital. Many of these are useless for my purpose, being hopelessly disorganised; but many others show similar changes to those found in the three recorded, the later being chosen because they present a well-marked gradation, and further, emphasise the main causes of otherwise conflicting results.

I propose, first, to review the rather scanty literature upon the subject. A vast amount of work has been done upon corneal wounds, but wounds of the other coats of the eye have received but meagre attention. This is the more remarkable in that the conditions present in the eye afford, in many ways, a very specially favourable field for settling some moot points in general pathology, apart from their specific interest to ophthalmologists. This fact has been fully recognised in dealing with the cornea and anterior chamber, the latter, of course, particularly from the bacteriological point of view. It is none the less patent in dealing with the sclera, choroid, retina and vitreous, all of which present special characteristics which enable us to exclude various contingencies which vitiate experiments upon other parts of the body.

Secondly, I propose to describe in detail the actual lesions produced in the experiments upon monkeys, and the

clinical and pathological notes of the lesions produced by accident in man.

Finally, I shall gather together, discuss and criticise the data obtained, adding a few remarks showing the bearing of some of the results upon the pathology of so-called "retinitis proliferans."

Historical.

As regards wounds of the *retina*, only one monograph of importance has been published. Doubtless many facts of importance are published incidently in the records of the pathological lesions occurring in human eyes, but these are invariably so vitiated by concomitant factors that they possess little importance unless controlled by experimental results. The same criticism would, of course, apply to the three pathological cases reported in this paper, were they not controlled by the experimental investigations.

The paper referred to is one by Tepljaschin,[1] of Kasan, published in 1894, on "The Histological Changes in the Retina after experimental Injury." This is an exhaustive paper on the subject, and some of the deductions drawn by the author from his preparations are so at variance with current pathological ideas, and my own conclusions, that it requires somewhat detailed consideration.

The experiments were made upon rabbits, "not less than 50 experiments" having been performed. An attempt was made to wound the retina without injuring the choroid and sclera: it is improbable that this was effected in many, if any, of the cases. A discission needle was passed through the coats of the globe 5—6 mm. behind the margin of the cornea, and a linear wound made in the retina on the opposite side. Perforating wounds were also examined, made by a Beer or Graefe knife inserted 4 mm. behind the corneal margin, a meridional section of 4—5 mm. being made, Finally, in other cases the retina was cauterised, as in the experiments of Roth[2] and Baquis,[3] by an electro-cautery thrust into the globe as far as possible behind the limbus.

The animals were chloroformed to death at intervals after the operation varying from 22 hours to 285 days. For vertical sections of the retina, the tissue was fixed in Flemming's weak or strong osmic-chromic-acetic mixture, and cut in celloidin. Flat preparations were stained by a modification of Ehrlich's *intra vitam* methylene blue method.

Perforating wounds, and those produced by cauterisation cause an increase of the wandering calls in the vitreous, commencing on the 2nd day and reaching a maximum on the 4th or 5th. This is most marked in the immediate vicinity of the wound, but extends for varying depths into the vitreous. Are these cells entirely due to emigration from the surrounding blood-vessels, or are they, at least in part, due to division of the normal wandering cells of the vitreous? H. Pagenstecher,[4] from experiments in which a foreign body was introduced into the vitreous, agrees with the majority of authors in regarding them as due to emigration. Hebb and Brailey,[5] on the other hand, in a paper on "Suppurative Hyalitis," "deduce the general conclusion that the migration of the colourless blood-corpuscles bears no part in these cases of suppuration of the vitreous body." Haensell,[6] from experimental data, holds the intermediate position regarding the increase to emigration from the retinal and choroidal vessels, supplemented by proliferation of the normal cells found in the vitreous. Potjechin[7] ascribes the primary increase to emigration, and subsequent increase on stronger or longer inflammation to proliferation. Wedl and Bock[8] figure vitreous cells in different stages of direct cell-division, and the possibility of simple cell-division in leucocytes is proved by the researches of Bizzozzero, Ranvier, Klein, Stricker, Flemming, Lawdowsky and others. That they can also increase by karyokinesis is shown by Peremeschko, Kultschizky, Marchand, Metschnikow, Schpronk and Flemming. Further, Arnold describes increase of leucocytes by so-called "fragmentation" (an appearance similar, if not identical, with that of the ordinary polymorphonuclear cell). Tepljaschin figures examples of each

mode of cell-division, from leucocytes found in the vitreous on the 4th day after operation.

Regressive metamorphoses occur in some cells even on the 1st day after operation, and these become more and more patent as time goes on, so that after the 4th or 5th day, when the leucocytes tend to decrease in numbers, they become predominant. The cells now show nuclei with deeply stained (with safranin) granules of varying shape and size, or the nuclei are broken up into small deeply stained particles. These are doubtless forms of nuclear débris from dead leucocytes (karyorhexis, karyolysis). Other cells show vacuoles; others again, fatty degeneration.

From the 4th to 5th day some of the leucocytes become grouped around new formed fibrous tissue, which fills the perforating wound. Karyokinetic figures are seen amongst these.

Sooner or later the inner surface of the retina (internal limiting membrane) becomes covered with flattened cells with well-marked oval nuclei. These are endothelial cells; "in flat preparations, stained with methylene blue, they are seen to be derived from wandering cells." (Tepljaschin). This appearance is familiar in pathological human eyes in many conditions, *e.g.*, chronic retinitis (Iwanoff),[9] shrunken vitreous (Duke Carl Theodor)[10], detached retina (Nordenson),[11] intraocular cysticercus (Dolina),[12] &c.

In later stages, a lamina of connective tissue often forms over the inner surface of the retina, "which is so intimately bound up with the retina, that the radial supporting fibres of the latter (*i.e.*, Müller's fibres) pass over into this lamina and take part in its formation." (Tepljaschin.) Similar structures are also seen in pathological human eyes, where they are known as retintis proliferans (Manz),[13] due, according to Leber,[14] to hæmorrhages. The question arises, can these fibrous tissue developments arise from wandering cells, or are the elements of other tissues necessary to their formation?

Much controversy has taken place in the realm of general

pathology as to this question, and, whilst the transition of leucocytes into granulation tissue was formerly admitted, much doubt has been cast upon it in recent years, dating especially from the opinions expressed by Ziegler, Marchand, Grawitz, and others at the 10th International Medical Congress in Berlin, in 1890. (Sherrington and Ballance* in the previous year published an important paper in which they practically proved that the new fibrous tissue cells do not arise from leucocytes, but from plasma cells which invade the injured area immediately after the leucocytic invasion. These plasma cells or fibroblasts are derived from those normally present in mesoblastic connective tissues. The method used was the examination of the films of tissue invading the space (1/20 mm. thick) between two cover-glasses, which had been embedded in the subcutaneous tissue of living rabbits or guinea-pigs for varying lengths of time from 4 hours to 18 days.)

As regards the eye, H. Pagenstecher's experiments were considered by him to confirm the then accepted view. Tepljaschin, using the methylene-blue method, lays great stress upon the fact that the leucocytes take up this stain, whilst the embryonic connective tissue cells do not. Many of the former are seen to arrange themselves in layers and become elongated, spindle or star-shaped. They thus approximate the form of new fibrous tissue cells. The same phenomenon was found to occur in a case of cyclitis; hence he concludes that "wandering cells of the vitreous can, in the latest stages after injury of the eye, and also under the influence of chronic inflammatory processes in the uveal tract, become transformed into fixed cell-elements."

The behaviour in the blood-vessels is next considered. In the rabbit, the large retinal blood-vessels lie on the inner side of the internal limiting membrane, the capillary vessels passing through it but penetrating only into the nerve-fibre layer (Krause). Injury of the larger vessels was avoided in the experiments. A few days after the operation,

* Sherrington and Balance, Jour. of Physiology, vol. x, 1889, p. 550.

proliferation of the endothelium of the capillaries by indirect division was observed. In later stages, large, flat, monocular cells, with long thin processes, were seen in the immediate neighbourhood of the vessels, apparently directly connected with their walls. Such cells are unknown in normal adult rabbits, but are found in fishes and frogs (Hans Virchow, Zimmerman), and also in the embryo (R. Virchow, Kölliker, Lieberkuhn). From flat preparations, stained with methylene-blue, Tepljaschin concludes that we have here "so-called vessel-forming cells and especially an intracellular process of new vessel-formation, like that described in embryonic development and in inflammatory processes by Ranvier, Ziegler, &c." New vessels were not formed in the vitreous except in the case of the masses of granulation tissue filling perforating wounds.

In the early stages, general retinal œdema was seen, most marked around the ganglion cells and the outer cells of the inner nuclear layer. This was seldom observed three or four weeks after operation, and was absent after one to two months.

In the nerve-fibre layer many varicose nerve-fibres were found, like those described by H. Müller[15] as characteristic of albuminuric retinitis, but since discovered in other conditions. They have been investigated by Roth and Baquis. They are found from 22 hours after injury, and occur both peripherally and centrally to the wound, but earlier centrally the nearer the wound to the papilla. On the peripheral side of the wound they occur later than on the central side, and they also appear later the nearer the wound to the papilla. The cut ends of the nerve-fibres swell up on the central side, but this does not occur so much upon the peripheral side; hence the former are globular, the latter club-shaped. Various hypotheses have been advanced to account for the varicose condition, such as sclerosis of the nerve fibres (Virchow), hyaline degeneration (Recklinghausen), hypertrophy of the nerve-fibres (H. Müller), hæmorrhage due to albuminuria (Berlin), post-mortem changes (Dogiel), &c.

(Virchow's view is probably the correct one: the phenomenon may even be due to an extremely fine myelin sheath, such as is known to be present on some nerve fibres formerly described as non-medullated. It is likely that a naked axis cylinder does not exist, except perhaps in the small ramifications at the terminal synapse. If this view be correct, the varicosity would be anticipated by analogy with other degenerating medullated fibres. The early onset of varicosity on the peripheral side when the injury is near the ganglion cell from which the axon springs is probably due to shock to the cell, and is analogous to the presence of Marchi degeneration in the central ends of certain motor nerves, *e.g.*, hypoglossal, when the nerve is forcibly torn out, a result which does not follow if the nerve is merely cut through. The shock to the cell is apparently always sufficient in the case of retinal injury to lead to ultimate degeneration of the part still attached to the ganglion cell, but is less when the wound is near the papilla, and therefore presumably farther from the cell. This extreme susceptibility of the retinal ganglion cells may also possibly account for the optic atrophy in cases of injury to the optic nerve in the optic foramen, &c. (*e.g.*, cases of fractured base), a degeneration which is difficult to reconcile with the general Wallerian law. At the same time the presence of efferent fibres in the optic nerve must be taken into account, and also the possibility that they are more numerous than has been hitherto suspected*). No regeneration of nerve fibres was observed, although the club-shaped extremity of the part united to the cell of origin is indicative of continued, if lowered, vitality. (Such a result was to be expected. The axons of the ganglion cells are analogous to true intraspinal axons, and on no reasonable hypothesis to spinal dorsal root fibres, as has sometimes been stated. These

* See my paper on Degenerations following Lesions of the Retina, Brain, vol. xxv, 1902. These questions are discussed here because they require special methods for their elucidation, and they do not come within the immediate purview of this paper.

latter are represented by intra-retinal fibres,—either those of the inner nuclear layer, or those of the outer nuclear layer, but *which* it is difficult to say.* The ganglion cell fibres belong, therefore, strictly speaking, to the central nervous system, and such, as is well known, never regenerate). In preparations fixed by Flemming's solution, black droplets were observed amongst the fibres, due to broken up myelin. (These doubtless represented some medullated fibres, which occur in abundance in the rabbit's retina. They cannot, therefore, be regarded as evidence of a fine myelin sheath to so-called "non-medullated fibres.") Leucocytes were oberved in large numbers around the degenerating fibres, distinguished from fixed cells by the fact that they stained with methylene blue. In late stages (194 days) ganglion cells are found only near the ora serrata on the peripheral side of the wound, whilst, still later (285 days), both ganglion cells and nerve fibres had entirely disappeared peripherally. In both these cases the nerve cells of the inner nuclear layer were intact, though they formed a thinner layer, due probably to the atrophy of the ganglion cell processes.

In the ganglion cell layer, the changes were essentially degenerative, but appearances of cell proliferation by karyokinesis were also present. Baquis observed them also, but attributed them to a simple reaction to irritation, since they were absent after seven days. Tepljaschin confirms this view, though he found many as late as the 15th day (most from 4th to 5th day). They were mostly in the monaster stage, and showed concomitant signs of degeneration, *e.g.*, fatty degeneration, vacuolation (hydropic degeneration), &c.

The changes in the inner reticular layer were principally due to œdema, such as swelling of the fibres and invasion by wandering cells.

The changes in the inner nuclear layer resembled those in the ganglion cell layer, but there were more mitotic

* See my paper on Toxic Amblyopias, Ophth. Review, July, 1901; or Barker's Nervous System, p. 541.

figures, belonging probably, according to Tepljaschin, to Müller's fibres, though some undoubtedly belonged to the nerve cells. Monasters occurred most frequently, and were also accompanied by degenerative changes. Especially noteworthy was the hyaline degeneration of some cells, similar to that described by Oeller[16] in Amblyopia saturnina, Hess[17] in naphthalin poisoning in rabbits, &c. It occurs chiefly in the outer row of cells, *i.e.*, where nutrition from the retinal vessels (in the rabbit) stops.

The outer reticular or inter-nuclear layer is somewhat atrophied, owing to the disappearance of the processes of the degenerating nuclear layer cells: œdema is associated with the atrophy.

In the outer nuclear layer, the nuclei are separated by œdema, the normally visible Müller's fibres being now visible, with their oval, faintly staining nuclei, often showing karyokinetic figures. The latter occur earlier here than elsewhere (2nd day, being absent after the 2nd week). Nuclear division of the nerve cells is only seen a short distance from the wound (4th to 6th day). After the 6th day some of these, situated in parts where destruction is limited to the rods and cones, resemble embryonic forms of these structures. The nucleus sends out a fine process, ending in a knob, through the external limiting membrane, the process being covered by a thin layer of cytoplasm. These resemble the inner limbs of cones, with ellipsoidal bodies (Kostenitsch). The nuclear chromatin of many cells in this layer shows degenerative changes, being broken up; so that later the layer becomes thin by destruction of the cells, débris of which is carried into other layers of the retina.

The rods and cones are either destroyed or in extremely degenerated condition, their place being taken by a homogeneous exudate. In places, however, elongated rods and cones were observed, such as have been described by Nettleship[18] in secondary glaucoma, Leber in glioma, Alt[19] albuminuric retinitis, Nordenson in detached retina, &c.

The pigment epithelium is separated from the rest of the

retina by exudate. The cells show fatty degeneration and vacuoles, though some show mitotic figures.

As regards wounds of the *choroid*, a few remarks are found in the same paper by Tepljaschin. The changes are essentially inflammatory and reparative. The tissues are separated by serous exudate and infiltrated by wandering cells, whilst karyokinetic figures form a distinct feature in vertical sections. They were principally "internal to the chorio-capillaris," in the layer described as the musculus chorioideæ in rabbits by Hällsten and Tigerstedt. Karyokinetic figures were also seen in the cells of the muscular layer of the walls of the large choroidal vessels.

The most important researches upon the healing of wounds of the *sclerotic* are those of Krückmann,[20] Franke, [1] and Stoewer.[22] The two latter operated upon rabbits, the former examining wounds of a duration of 4, 8, 12, 24 hours, and every other day up to 18 days. Krückmann paid most attention to the changes during the first 24 hours, but also continued the investigation up to 120 days: he experimented upon guinea-pigs, rats, cats, and dogs as well as rabbits.

According to Krückmann, the invasion of the wound and neighbouring parts by leucocytes is the prominent feature 4 to 6 hours after the operation. These leucocytes are poured fourth from all the blood vessels available, viz., those of the episclera, choroid, and such sclerotic vessels as are to be found. They are carried from all sides into the wound, which they block, the groups being thickest along the edges. They penetrate into the dilated lymphatic channels between the scleral lamellæ ("Infiltrations-Scleritis" of Alt). The meshes of the suprachoroidea are also packed with them. Their function is largely a phagocytic one, directed to the removal of the parts injured beyond repair. The edges of the wound become smooth and glazed, so that the process differs in no essential from that which occurs in other parts of the body. In the course of the 2nd day the leucocytes

begin to disappear, their duty as scavengers having been accomplished.

Franke describes the new formation of round and spindle-shaped cells during this period, by a process of amitotic division. (The need for rapid increase of cells in all acute inflammatory processes results in division by fission, gemmation, &c., ordinary karyokinesis being apparently too slow. This is seen *par excellence* in the cornea.) It is not surprising that some increase of the cells present should occur at this early stage, but it is probable that most of them are leucocytes (Krückmann, Stoewer), and that the process described by Franke plays a very subsidiary part.

Franke observed the evidences of karyokinesis after 43 hours, which agrees well with the time assigned by Krückmann for the commencement of the second stage, viz., the process of repair. By this time the detritus and perhaps much of the fibrinous coagulum which formed a large part of the inflammatory exudate have been removed by the agency of the leucocytes, and these themselves have in turn largely disappeared. It is now that the activity of the fixed tissue elements forms a leading feature, and, as in other parts of the body, the blood vessels are the most important. In the episcleral tissue and conjunctiva, in the interstices of the neighbouring muscle fibres, and in the choroid, many cells are seen dividing by mitosis, but especially those belonging to the adventitia of the blood vessels. These (probably including the plasma cells) proliferate rapidly, forming spindle-shaped cells, *i.e.*, embryonic fibrous tissue, which invade the wound area, accompanied by new formed capillaries. Many types of cells are seen in many stages of proliferation, but besides those already mentioned endothelial cells are very prominent. The scanty scleral corpuscles are not inactive, but also proliferate. They do not appear, however, to invade the wound area until a later period. The vitreous has a purely passive part in the process, except that it becomes fibrillated (Franke). The retina is also passive, becoming glued down to the choroid

and locally degenerated. (The retinal vessels probably play a small part in common with those of the choroid.)

Düffing[25] examined an eye microscopically four weeks after a double perforating wound of the sclerotic by a small saw. He found that the fibres of the scar now had the same direction as those of the normal sclera, and passed imperceptibly into the surrounding normal tissue. This result was confirmed by Franke's experiments on rabbits. (It is at this stage in all probability that the normal sclerotic corpuscles manifest their influence. They probably reproduce themselves slowly and partially replace the scar tissue. It is not unlikely, too, that they influence the polarity and character of the newly formed fibrous tissue, tending to gradually mould the new cells to their own type. Such an "infective" influence of one cell upon its neighbours is not uncommon in many pathological conditions (*e.g.* new growths), but where the injured normal cells are of a high degree of differentiation, the process usually stops short at a lower grade, as is seen in the latest stages of scars in the cornea. These become more allied to sclerotic cells than to corneal corpuscles, possibly a form of atavism. In the case of the sclerotic itself, the lowly grade of cell is more readily approximated by the new formed corpuscles.)

Experiments on Monkeys.

Method.—In my experiments, operations were performed upon six monkeys. The pupils having been previously well dilated by the instillation of atropine, the animal was anæsthetised with ether. The fundus was examined ophthalmoscopically to see that it was normal, and the conjunctival sac was washed out with weak solution of perchloride of mercury. A speculum was inserted, and the eye fixed by fixation-forceps, held by an assistant. A Graefe's cataract knife was then introduced into the eye 3 or 4 mm. behind the corneo-scleral margin, at either the nasal or temporal side, thus avoiding dangerous injury to the ciliary body

and minimising injury to the retina. The knife was passed across the eye through the vitreous to the opposite side, and the retina wounded there to the required extent, the position of the point of the knife being in some cases observed by the ophthalmoscope (direct method). The ophthalmoscope was especially used in lesions near the disc or macula. In other cases, the wound was made without ophthalmoscopic examination, the slight resistance encountered being sufficient to show that the retina had been reached. The exact position and nature of the wound were subsequently determined by ophthalmoscopic examination, which was repeated after an interval of a few days, and also shortly before killing the animal. The lesions were all investigated macroscopically by a suitable section of the eye post-mortem, and were then prepared for microscopic examination.

There was usually no loss of vitreous during the operation, but a small bead escaped in two cases, without, however, causing any untoward result. The wound of entrance healed with great rapidity in all cases. There was slight hyperæmia of the conjunctiva for a day or two, but this quickly passed off, and the animal looked perfectly normal.

The monkeys were chloroformed to death from a fortnight to three weeks after the injury.

The primary object of the experiments was to trace the degenerations in the optic nerves, chiasma, and tracts. These results are recorded elsewhere.

The eyes were hardened in 10 per cent. formol, frozen, and bisected in the direction most suitable for examination of the particular lesion—generally equatorially. The part to be investigated was passed through alcohols, embedded in celloidin, and cut.

Experiment I. Rhæsus. Right Eye.

16.x.01. A small lesion, about 2 mm. long, in a direction tangential to the disc, was made near the equator in the lower nasal quadrant. There was a little hæmorrhage; the

area of pigmentary degeneration was very little larger than the lesion.

31.x.01. Killed.

Microscopical Examination.—Cornea: a.c., iris and lens normal. The posterior wound passes through the retina and choroid, and just through the sclerotic. The edges of the sclerotic are turned inwards and separated by a plug of new formed fibrous tissue derived from the episcleral tissue. This forms the centre of a mass of more fully developed connective-tissue which extends far into the vitreous (Fig. 1). Anteriorly it spreads out and branches, long fine filaments spreading out as far as the ciliary body. The ciliary body is not inflamed. The non-pigmented cubical cells of the pars ciliaris retinæ are irregularly elongated, and appear in places to have definitely proliferated. The inner ends are in contact with extremely delicate filaments made up of long spindle-shaped and branching-cells, the filaments lying almost parallel to the surface, sweeping round in a wide curve towards the lens, and then back to be lost in the mass of tissue springing from the wound. Scattered leucocytes, few in number, lie upon the pars ciliaris retinæ and pervade the new formed tissue. They are quite typical in their appearance, though they belong to different kinds of white cells, but no appearances in any degree suggestive of transition into spindle-shaped cells are seen. Many of the leucocytes look perfectly normal, many others are obviously disintegrating. Many cells contain pigment granules in their cytoplasm (Fig. 2), and in places free pigment granules are abundant (Fig. 3). Most of the pigment-containing cells are undoubtedly leucocytes, and none can be definitely stated to be true pigment cells (Fig. 2.) They and the free granules are especially abundant near red blood corpuscles, of which there are comparatively few in this eye, no large vessel having been wounded in this experiment. Where they occur they are usually arranged in layers between the parallel fibrous filaments: there is no evidence of new capillary formation in the anterior part of the eye.

FIG. 1. × 25.

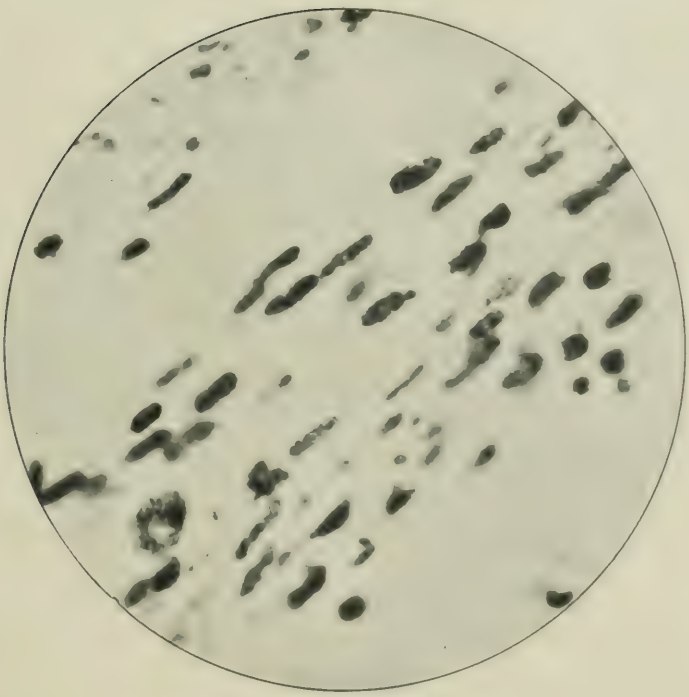

FIG. 2. × 300.

PARSONS—Wounds.

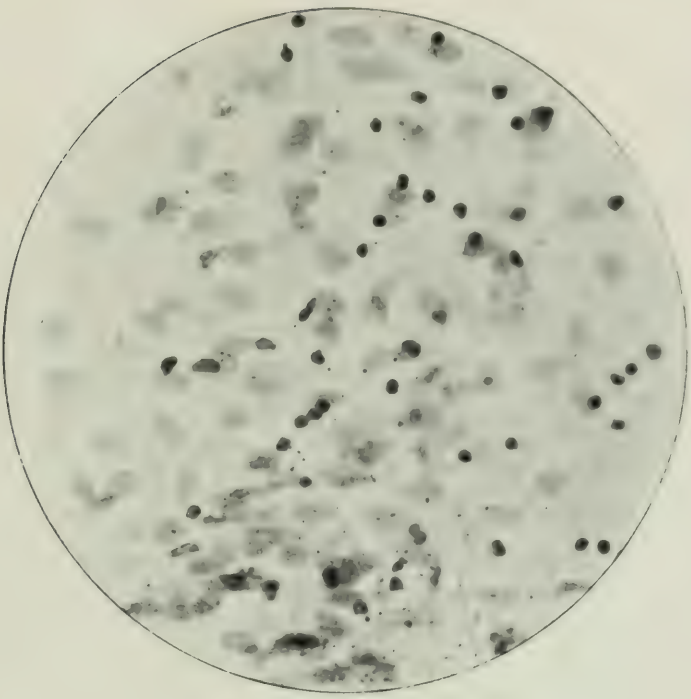

FIG. 3. × 300.

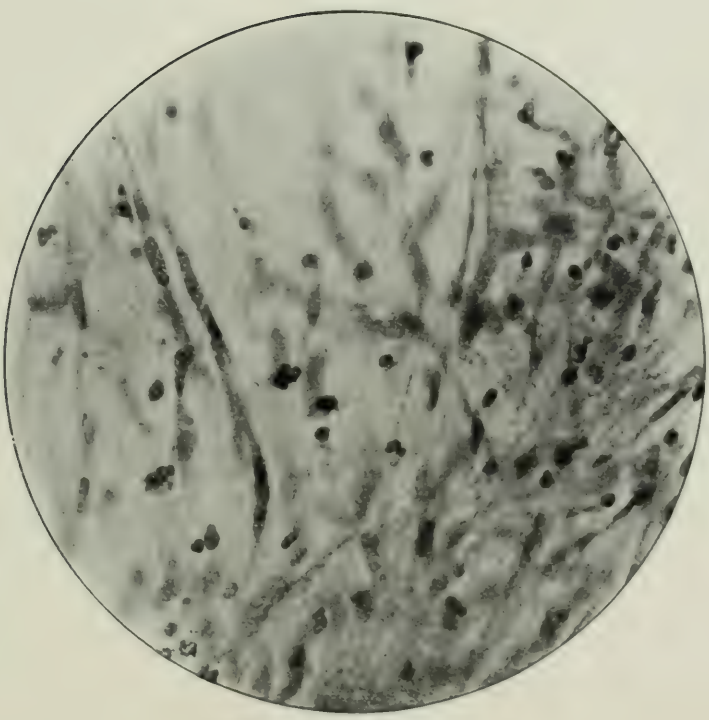

FIG. 4. × 300.

PARSONS—Wounds.

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Considering next the main mass of tissue filling the wound, the central core, pyramidal in shape with the apex forward, derived from the episcleral tissue, contains more leucocytes than the remainder. In the centre is a widely-dilated, thin walled new vessel, containing red corpuscles. The leucocytes and some pigment cells are most marked near this vessel. Covering this pyramidal process of episcleral tissue are the in-turned edges of the sclerotic and choroid, outside which the very slightly altered retina is adherent. (The general detachment of the retina in the figure is due to the preparation of the specimen, and was not present when the eye was bisected.) The edges of the sclerotic can be made out in the sections, though they are rounded off and capped with fibrous tissue, which is furthest developed in this situation. The choroid spreads out and is lost in a more highly nucleated connective tissue, amongst which pigment cells are seen: these dwindle rapidly as the tissue is traced inwards. The main mass spreading out into the vitreous consists of closely packed laminæ, made up of imbricated spindle-shaped and branched cells, with long oval nuclei. The tissue is most organised at the base, spreading out into processes which extend far into the vitreous and consist of looser tissue of the same kind. Here the shape of the cells is most easily demonstrated (Fig. 4). Interspersed amongst the embryonic connective tissue cells are a comparatively small number of leucocytes, recognised by the deeply stained round nuclei, and a few endothelial cells. The latter are recognised by their large oval (but little elongated) faintly staining nuclei, with deeply stained nucleoli. They are seen with difficulty in the base of the mass, where the tissue is very compact, but deeper in the vitreous they become prominent, and in places are extremely numerous (Fig. 3).

The retina still retains its layers intact, and is only slightly altered even close to the wound. Here there is slight œdema of the inter-nuclear layer. The pigment epithelium, rods and cones, and the nuclear layers are normal.

The nerve-fibre layer and the vessels appear normal, except peripheral to the wound where many of the ganglion cells have degenerated, and the nerve-fibre layer contains many round cells. These are not conspicuous in other parts of the retina.

The choroid is apparently normal except close to the wound. It is deeply pigmented in the monkey, and it is impossible to make out its finer structure without bleaching, which would have spoiled more important structures. Near the wound there is an enormous development of new fibrous tissue growing directly into the proliferating mass, which is evidently largely derived from this coat. There is no evidence that the sclerotic takes any part in this process, but this point is better demonstrated in other specimens.

The optic nerve showed degeneration when examined by the Marchi method. Degenerated fibres were scattered in small numbers over the whole transverse section, but there was a well-marked aggregation on the nasal side corresponding to the retinal wound. (The degenerations in all these monkeys are described in detail elsewhere.)[21]

Experiment II. Jew. Left Eye.

30.x.01.—A rather larger lesion, about 3 mm. long, was made in a direction tangential to the disc a little in front of the equator in the lower temporal quadrant of the retina. There was practically no hæmorrhage, and the pigmentary degeneration was limited to the lesion.

18.xi.01.—Killed.

Microscopical Examination.—Cornea, a.c., iris and lens normal. Part of the wound through which the knife entered the eye is seen in Fig. 5. It will be noticed that it passes through the posterior part of the ciliary body, and anterior to the retina proper. It is filled with new fibrous tissue very similar to that described in Experiment I, but projecting only a short distance into the vitreous. This tissue is principally derived from the subconjunctival or episcleral tissue,

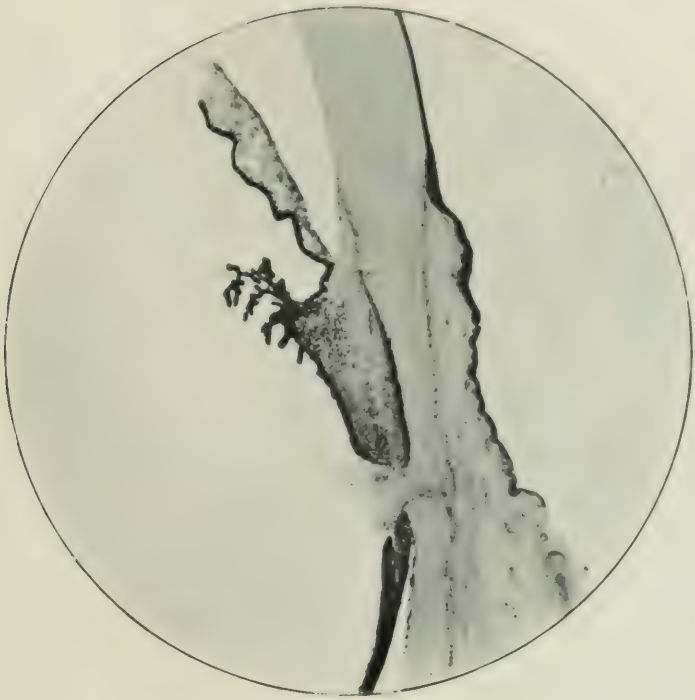

Fig. 5. × 10.

Fig. 6. × 60.

Parsons—Wounds.

as is proved by other sections, and in much less degree from the uveal tract, as is seen in Fig. 5. The pars ciliaris retinæ plainly takes no part in the process at all, except that the pigment cells of this layer sometimes show proliferation, but only in slight degree. The sclerotic, too, is absolutely inert, the edges being clean cut, though slightly rounded off, and the new cells running almost entirely at right angles to them. As mentioned, the ciliary body provides fibroblasts for a modicum of the scar tissue, and some proliferation of its pigment cells also occurs. Pigment is found in small quantities irregularly scattered through the scar, mostly as free granules, some in leucocytes, and some—the smallest part—in pigment cells, most of which are of uveal origin, though some appear to be retinal. It is clear that this lesion produces a minimum of disturbance to the eye, and is probably far less than that produced by the electro-cautery.

The posterior wound, though long (3 mm.), is limited to the retina and choroid, the sclerotic being absolutely intact (Fig. 6). Possibly only the retina was wounded, the choroidal atrophy being secondary: this at least is probable from the minute amount of hæmorrhage, and the limitation of the pigmentary disturbance. It is seen that the retina and choroid are replaced by a well-developed scar of fibrous tissue. The amount of organisation is greater than in Experiment I, on account of the smaller extent and severity of the lesion, and also on account of the longer interval before death (19 as compared with 15 days). The retina and choroid are seen to be closely matted together at the edges of the wound, so that the retina remains adherent here after the processes necessary for microscopical examination. The scar tissue consists of layers of imbricated spindle-shaped cells closely packed together, and having long thin, deeply staining, oval nuclei. They are directly continuous on each side of the choroid, and the retina is merely adherent to them, and seems to have had absolutely nothing to do with their formation. This is very obvious in the stained specimens, where the difference in their avidity for eosin is very

marked, the retina being deep red, and the new cell protoplasm being scarcely stained. The choroidal pigment invades the edges only of the scar, very little in front (Fig. 7), rather farther behind. A few free pigment granules are seen, but beyond these constituents no others can be made out. On the vitreous surface are a few scarcely altered red-blood corpuscles, and a very few leucocytes, one or two of which contain pigment granules.

The retina and choroid were *in situ*, and generally normal. At the posterior edge of the wound the retina is disorganised for a small fraction of a millimetre, and even here there is a normal looking ganglion cell, and the nuclear layers are easily distinguishable, only the rods and cones and the fibre layers having in large part disappeared. Peripherally to the wound, on the other hand, the retina is very much atrophied, the nuclear layers having run together for a considerable distance. Here there are scarcely any ganglion cells, and the reticular layers are degenerated, though the rods and cones are intact over the mid area between the scar and the ora serrata. At the same time, though atrophic, there is no evidence of present or past inflammation. Possibly some of the nuclei may represent leucocytes, but if so, they are not to be distinguished from the nerve cells of the nuclear layers, and there is no new-formed connective tissue.

The choroid was *in situ*, and normal, except at the edges of the wound and over its area.

The optic nerve showed specific degeneration.

Experiment III. Bonnet. Left Eye.

20.xi.01.—A very small lesion was made upon the temporal side near the equator, just below the horizontal meridian. There was no bleeding. Macroscopically there was only a white dot at the site of the lesion.

7.xii.01—Killed.

Microscopical Examination.—Cornea, a.c., iris and lens

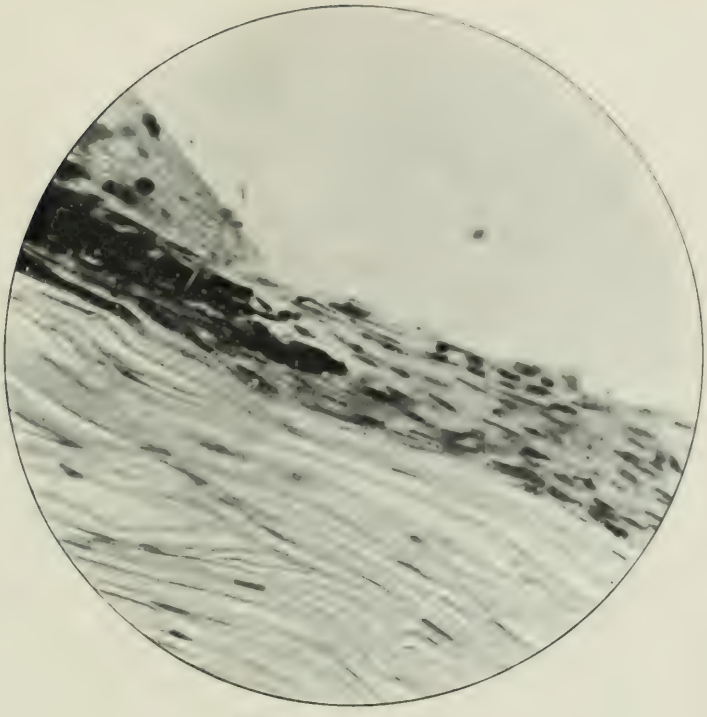

FIG. 7. × 300.

FIG. 8. × 120.

PARSONS—Wounds.

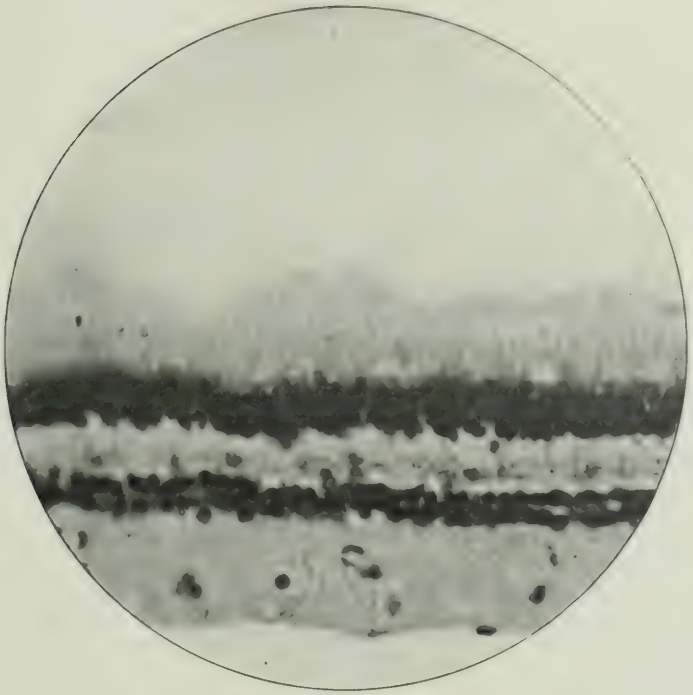

Fig. 9. × 300.

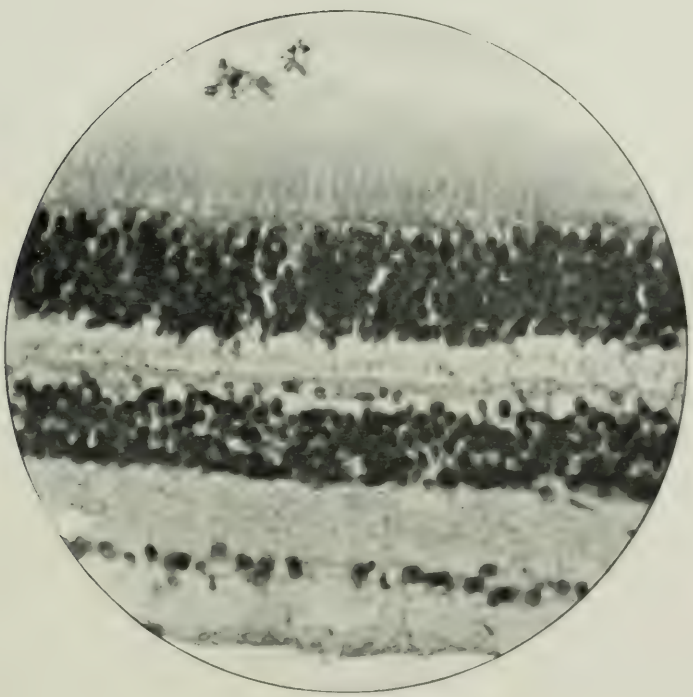

Fig. 10. × 300.

Parsons—Wounds.

normal. The posterior wound is an extremely small lesion (Fig. 8), involving only the retina and choroid, though probably the inner fibres of the sclerotic were injured: it was certainly not perforated. Microscopically, the lesion resembles that in Experiment II very closely, but rather less scar tissue has been formed. This is of exactly the same type, but is thinner and more pigmented. It is clearly derived from the choroid, though it spreads out at each end and forms two forks, into which the adherent edges of the retina fit.

The retina was *in situ*, and was normal posteriorly up to the adherent edge, which is broader than in II. Here the nuclear layers are clearly defined up to the extreme edge, and some rods and cones can be made out even in the adherent part. Peripherally to the wound the layers are also distinct and apparently normal, with the exception of the extreme sparcity of ganglion cells. Figs. 9 and 10 represent the retina centrally and peripherally of the wound respectively, and the great diminution in the ganglion cells is very obvious.

The choroid was *in situ*, and normal, except that it was atrophied at the wound.

The optic nerve showed specific degeneration.

Experiment IV. Rhæsus. Right Eye.

13.i.02.—The lesion was larger, about 3 mm, long, in this retina. It was on the temporal side, a little behind the equator, and extended above and below the horizontal meridian. There was slight hæmorrhage, and post-mortem there was an oval area of pigmentary degeneration, with axes of about 4 mm. by 3·5 mm. The retina was firmly adherent to the choroid over this area.

1.ii.02.—Killed.

Microscopical Examination.—Cornea, a.c., iris and lens normal. The posterior wound is particularly interesting: the posterior part closely resembles that in Experiment II,

and it may be noted that the period from injury to death is the same (19 days). The anterior part again closely resembles that in Experiment III, whilst between the two we have an area presenting new features. The scar tissue in the posterior part is exactly the same as that shown in Figs. 6 and 7, except that it extends as a thick layer for a short distance over the retina, which is clearly separable from it, though the retina is much atrophied here and represented only by a layer of persistent nuclei of one of the nuclear layers. In the anterior part there are more layers of fibrous tissue than in Fig. 8, and they are rather better organised, facts which are consistent with the slightly longer period (19 days as compared with 17), and the larger, and, therefore, severer wound. Moreover, there is more pigment proliferation : and near the retinal margin some rounder, more faintly staining nuclei, reminiscent of endothelial cells, are seen amongst the long oval deeply-staining nuclei. The central part is noteworthy, in that there is a considerable patch of atrophic retina persisting (Fig. 12). The choroid has almost disappeared here, and the retina lies almost upon the sclerotic. It is represented by nuclei of one or both of the nuclear layers, varying from one or two to six or eight in thickness Upon these lies an eosin-staining fibrillated layer with no nuclei proper to itself, such nuclei as occur belonging undoubtedly to vagrant cells. Superficial to this, and separating the lower layers from the vitreous, is the continuation of the scar tissue, here only two or three cell-layers thick. Scattered through all these layers are pigment cells, some of which have all the characteristics of retinal pigment epithelium, and the condition of the retina resembles closely the condition found in many pathological human eyes. Some of the cells of the fibrous layer send off processes at right angles to the surface into the vitreous, especially over the degenerated retina. There are a few blood-corpuscles and one or two leucocytes, here and there upon the surface in the vitreous.

The retina was *in situ*. Posteriorly it is practically

FIG. 11. × 17.

FIG. 12. × 300.

PARSONS Wounds.

Bale & Danielsson, Ltd.

Fig. 13. × 25.

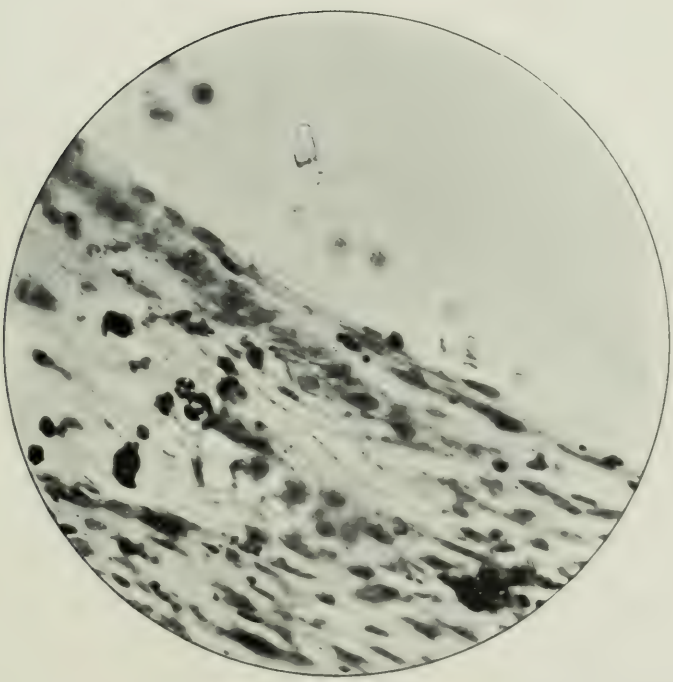

Fig. 14. × 300.

Parsons—Wounds.

Hazell & Innocent, Ltd.

normal, except close to the wound, where the changes are similar to those noted in other cases, except that here, too, we have evidences of proliferation and migration of the retinal pigment epithelium, some such cells being found close to the vitreous surface.

The choroid was *in situ*, and is also normal, except in the immediate vicinity of the wound. It would be difficult to affirm from the appearances in this case alone that the scar tissue was mainly derived from this coat, but in the light of the information obtained from other cases it is possible to see that such must have been the case. At any rate there is no evidence in this case against such a view.

The optic nerve showed specific degeneration.

Experiment V. Rhæsus. Left Eye.

14.i.02.—A small lesion was made between the macula and the disc, but at a slightly lower level. It was rather deep, injuring the choroid and sclera, and there was some hæmorrhage.

28.i.02.—The monkey died in the morning; it was dissected about two hours afterwards.

Microscopical Examination.—Cornea, a.c., iris and lens normal. Fig. 13 represents the wound of entry of the knife. The sections were cut eqatorially in this case, but exhibit the same features as in Fig. 5, only on a larger scale. The essential origin of the scar tissue from the episclera is well shown.

The posterior wound shows some characteristics which are less marked in the other cases. Over a small area the sclera has been completely divided; the cut edges, instead of turning inwards, have retracted slightly. The gap is filled with new fibrous tissue, which lies flat upon the normal episcleral tissue, the direction of the cells being, therefore, concentric with the curve of the globe, and not at right angles as in the other perforating wounds. Probably the scleral injury here was wider in extent but less severe, so that no

marked proliferation has taken place into the vitreous. The foundations of the new tissue are the same spindle cells as in other cases, but endothelial proliferation is more marked (Fig. 14). The best defined layer of new endothelial cells is found in this specimen, although it does not show very clearly in the figure. The main origin of the scar from choroid is indicated by the large amount of uveal pigment scattered about, but there are also many proliferated retinal epithelial cells. The episclera appears to be more passive than usual. The retina at the edges of the wound shows the usual type of degeneration, passing rapidly into more normal parts. The degeneration of ganglion cells, &c., peripheral to the wound is well marked. There is some hæmorrhage in the vitreous: but there are very few leucocytes present, and absorption without organisation is going on.

The optic nerve showed specific degeneration.

Experiment VI. Rhæsus. Left Eye.

25.i.02.—A lesion, 2 mm. long, was made in a direction down and out, starting 2 mm. down and out from the fovea centralis. There was considerable hæmorrhage into the vitreous.

15.ii.02.—Killed.

Microscopical Examination.—Cornea, a.c., iris and lens normal. The posterior wound penetrated all three layers Fig. 15 represents the appearance of sections of the wound. The cut edges of sclera are close together, the small space being filled with new tissue principally derived from the choroid, but partly from the episclera. There are many pigment cells in it. Only the cut edges of the retina appear to have suffered much—where they are bound down to the choroid and sclerotic. A stream of fibrous tissue passes forwards into a mass of red corpuscles lying in the vitreous. A few pigment cells, endothelial cells, and leucocytes are interspersed here and there. Organisation has proceeded farther than in any of the other cases in accordance with the

FIG. 15. × 28.

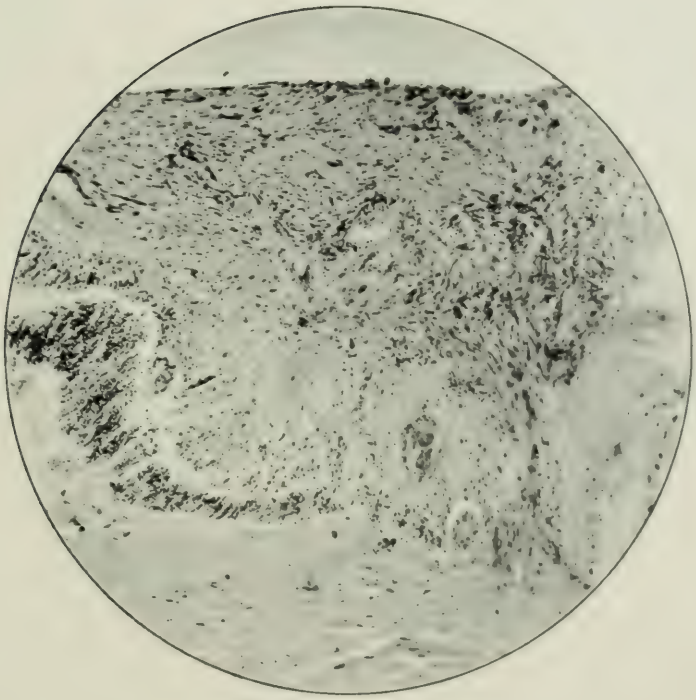

FIG. 16. × 60.

PARSONS—Wounds.

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longer interval after the wound. Masses of red corpuscles are found elsewhere in the vitreous, undergoing absorption, but with only small traces of organisation in the form of fibrous filaments in and around them. Other parts of the eye show remarkably little change.

The optic nerve showed specific degeneration.

Case I.

T. G. (6372), male, æt. 19, was admitted to the R.L.O.H. on 6.v.02, under Mr. Holmes Spicer. On the previous day he was struck by a piece of steel in the R. eye, whilst rivetting.

6.v.02.—*R. eye.* Much conjunctival and ciliary congestion. Small perforating wound of cornea on nasal side near periphery. Hypopyon. Lens probably wounded. Pupil irregular, dilated. White haze in vitreous; no red reflex. V. = p.l. T. −.

L. eye. V. = 6/6.

7.v.02.—Applied to Haab's magnet without result.

9.v.02.—R. excision.

Pathological Examination.—Eye hardened in 10 per cent. formol, frozen, and bisected horizontally through wound.

Macroscopic Examination.—Cornea — wound, with iris adherent. Lens—wounded, and drawn up to corneal wound. Vitreous—contains blood and leucocytes. A sector of steel, about 3 mm. long, was found deeply and firmly embedded in the upper nasal quadrant, a little behind the equator.

Microscopical Examination.—Cornea—slight œdema of epithelium and substantia propria; the wound shows considerable advance towards repair, being partially cicatrised. A.c.—good; angles and anterior surface of iris and lens covered with leucocytes; canal of Schlemm widely dilated and filled with blood. Iris—some iritis, but this is chiefly confined to vicinity of injury, and is less than might be expected. Lens—wounded; capsule torn and convoluted; lamellæ separated at site of injury by wedge-shaped plugs of leucocytes; there is very marked and well-advanced development of new fibrous tissue around the torn capsule in the neighbourhood of the injury. Interspersed amongst the new fibres are pigment cells and red blood corpuscles. Ciliary body — congested. Vitreous — contains red

corpuscles and leucocytes. Retina—looks practically normal except in the immediate neighbourhood of adhesion of f.b., but the vessels are dilated and filled with red corpuscles, and there is some round-celled infiltration of the adventitia. Choroid—normal, except at the same region, where it is enormously congested; even here, there is but little leucocytic infiltration. At the site of injury and lodgement of the f.b., the retina is adherent to the choroid, which is, in turn, knitted to the sclera. The actual bed of the f.b. is a mass of leucocytes and blood corpuscles lying on choroid, which is filled with round cells and pigment cells, the retina being quite destroyed here with the exception of a few vessels. (Fig. 17 is taken from this part.) A little peripherally, however, the change is very different, a well-marked capsule of fibrous tissue having already formed (Fig. 16). Quite a large mass of fibrous tissue has been formed, lying upon a small area of degenerated retina, which suddenly passes on each side into almost normal retina. The fibrous tissue has thin extensions on each side over the quasi-normal retina, gradually thinning off and disappearing. Many proliferated retinal pigment cells are caught in the meshes of the connective tissue. A coarse strand of fibrous tissue is seen passing from the choroid through the degenerated retina into the main mass, whilst the remainder of the tissue is directly continuous with the adventitia of the retinal vessels which have persisted in this part. These features are so marked as to leave little doubt that this new-formed fibrous tissue is directly derived, on the one hand from the choroid, and on the other from the adventitia of the retinal vessels. The choroid underlying this atrophic retinal patch itself consists of cicatricial tissue, passing on each side with but little gradation into the contiguous inflamed choroid.

Case II.

F. G. H. (6418), male, æt. 50, was admitted to the R.L.O.H. on 7.vii.02, under Mr. Lang. He had been struck in the L. eye on the same afternoon by a fragment of steel.

7.vii.02—*L. eye.* F.b. removed from between the lips of wound by forceps. There was a horizontal wound involving

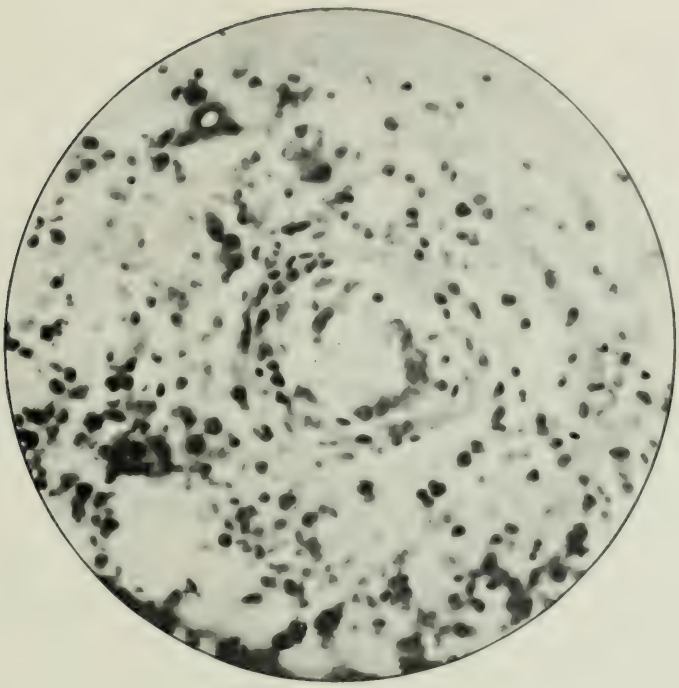

FIG. 17. × 300.

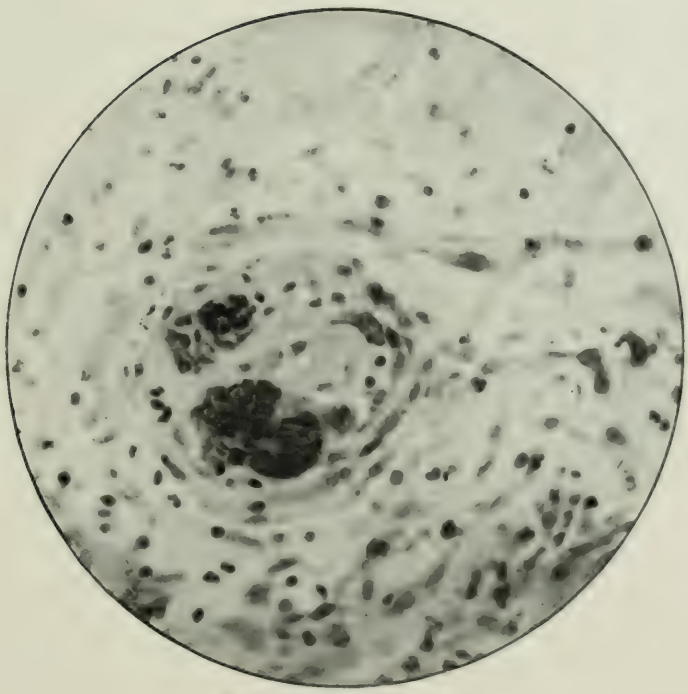

FIG. 18. × 300.

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both cornea and sclera, to the outer side, in the horizontal meridian. Aqueous—turbid: hyphæma. Iris—adherent to corneal wound; iritis.

R eye. V = 6/12 : c. +0·75 sph. = 6/5.

Haab's magnet was applied without result. Iridectomy of the adherent iris was performed.

The patient failed to present himself for X-ray examination.

14.vii.02. Hypopyon.

15.vii.02. L. excision.

Pathological Examination.—Eye hardened in 10 per cent. formol, frozen, and bisected horizontally through the wound.

Macroscopic Examination.—Corneo-scleral wound to outer side; lips infiltrated. Iris—coloboma outwards; iris elsewhere covered with lymph. Lens—traumatic cataract; lens drawn up to wound. Vitreous—adherent to wound; traversed by white filaments; some blood and pus. Retina, choroid and sclera—perforated near posterior pole, slightly external to macula; white filaments radiate forward from this spot to the anterior wound and the lens; others pass forwards from optic disc. There is a large fold of the retina between the disc and the wound, but it does not quite reach the latter.

Microscopical Examination.—Cornea — epithelium irregular near wound: œdema: infiltration of lips of wound, extending for a short distance around. A. c.—much exudate; angles full of leucocytes; canal of Schlemm dilated, and full of red corpuscles. Iris—acute iritis; hæmorrhages. Ciliary body—cyclitis; hæmorrhages; ciliary processes covered with leucocytes and fibrin. Lens—perforated, traumatic cataract; much swelling, especially at posterior wound of capsule; lamellæ separated by wedge-shaped plugs of leucocytes. Vitreous—full of fibrinous exudate, containing leucocytes and red corpuscles. Optic disc—slightly swollen (swelling doubtless diminished by dehydration during preparation); physiological cup filled with leucocytes, which also infiltrate the nerve anterior to the lamina cribrosa in large numbers. Optic nerve—slightly infiltrated; Retina—much congested and infiltrated; layers preserved intact; much folding between disc and wound. Choroid—congested and infiltrated, but less so than in many similar cases. At

the site of the posterior injury the retina and choroid are perforated, and the sclerotic is deeply wounded. The retinal vessels in the immediate neighbourhood survive best and show the best evidences of an almost abortive attempt at repair. Embryonic connective-tissue cells stream out from their adventitia into the surrounding mass of leucocytes (Fig. 18), fibrin and red corpuscles, but the fibrous element soon dwindles down and the main part of the wound is made up of polymorphonuclear leucocytes, phagocytes containing pigment granules, red blood-corpuscles, scattered pigment cells and fibrin (Fig. 19). There are comparatively few endothelial cells; and the connective tissue corpuscles extend but a short distance into the vitreous. A little farther from the wound the layers of the retina are intact, but are infiltrated with round cells, especially the nerve-fibre layer, which is crammed with polymorphonuclear leucocytes, and the inner nuclear layer in less degree. The choroid in this situation is similarly choked with round cells, but here they are almost entirely mononuclear. As is usual in scleral wounds, we find this tissue here almost inert. The edges are clean cut, and only slightly infiltrated, the space being filled with a plug of new fibrous tissue derived from the choroid, its continuity with this membrane being particularly evident.

Case III.

J. P. (6349), male, æt. 52, was admitted to the R.L.O.H. on 12.iii.02, under Mr. Gunn. On 3.iii.02, he was breaking a piece of iron with a hammer, when a piece flew up and struck his R. eye.

12.iii.02. *R. eye.* Scleral wound on nasal side, immediately behind limbus, about 3 mm. long, directed horizontally. Pupil eccentric and irregular; does not react: post. synechiæ. V = hand movements at 2 feet.

20.iii.02. *X-ray Report.*—"Two stereoscopic skiagrams show the presence of a piece of metal in the vitreous."

21.iii.02. Admitted an in-patient.

R. eye.—Much congested; scleral wound as above described. A. c. good. Atropine mydriasis; posterior synechiæ. Lens apparently clear. Hæmorrhage in vitreous: dull red reflex V = counts fingers at $\frac{1}{2}$ m.: projection good. Tn.

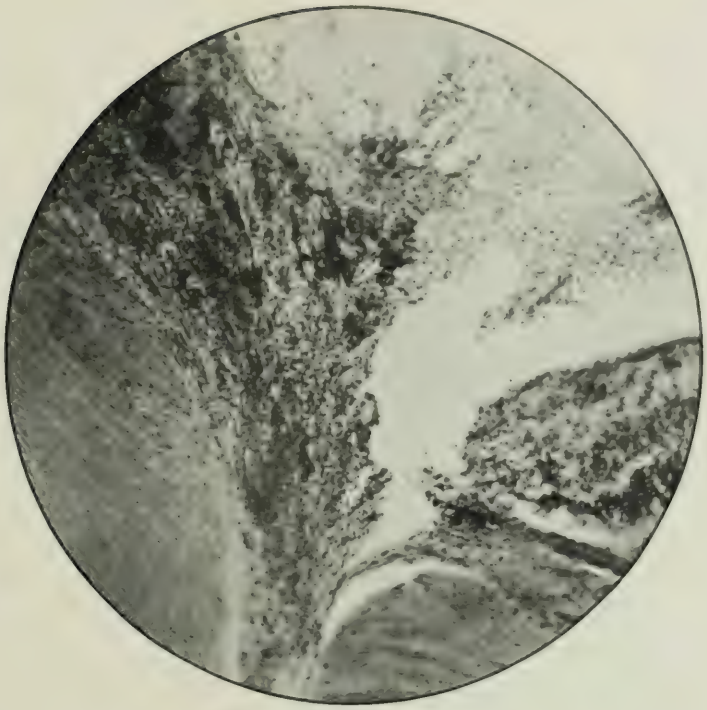

FIG. 19. × 60.

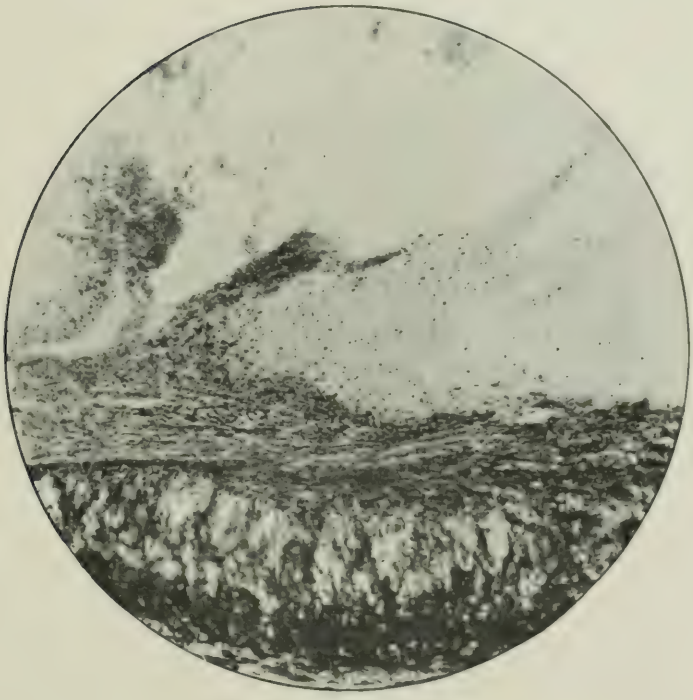

FIG. 20. × 60.

PARSONS—Wounds.

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L. eye. Cornea clear. Irritable pupil. V = 6 6 (?), illiterate. Tn.

22.iii.02. R. excision.

PATHOLOGICAL EXAMINATION.—Eye hardened in 10 per cent. formol, frozen and bisected sagittally.

Macroscopic Examination.—A small piece of steel was found firmly embedded in the posterior part of the ciliary body, just anterior to the ora serrata, in a direction downwards and very slightly inwards. There was a small amount of blood in the vitreous, but otherwise the eye looked healthy macroscopically.

Microscopical Examination.—Cornea—normal in greater part of area; slight infiltration and vascularisation of extreme periphery. Conjunctiva — much infiltrated and congested. A. c.—good; upper angle open, lower angle contains a few leucocytes in a fibrinous network; canal of Schlemm dilated, and full of blood. Iris—slight iritis, shown by a few leucocytes throughout stroma, and slight extravasation of blood at the extreme periphery into the stroma. Lens—normal. Ciliary body—acute cyclitis, most marked near site of f.b.: here the ciliary body is much congested, slightly infiltrated with round cells and covered by them. At the site of the wound there is a dense mass of leucocytes lying upon the altered pars ciliaris retinæ (Fig. 20). The leucocytes lie in a meshwork of fibrin, amongst which are a few strands of new-formed fibrous tissue. The striking feature is the intense leucocytic activity and the very slight effort at repair in the form of fibrous tissue development. In the posterior part of the main focus the choroid is much congested; and the retina is intensely infiltrated, pervaded by proliferated retinal epithelial cells, and shows marked connective-tissue hyperplasia. Retina —shows much inflammation and some degenerative changes throughout, these diminishing with distance from the main focus. Choroid—enormously congested everywhere, no gradation being noticeable. Vitreous—filled with scattered leucocytes.

REMARKS.

In considering these lesions there are certain fundamental points which must be continually borne in mind as being of cardinal importance in the process of repair. The first of

these is the fact that the experimental lesions were all absolutely aseptic: there was scarcely any reaction, and after a day or two the eyes looked quite normal; further, there was no trace of pus in the a.c. or vitreous. The pathological cases, on the other hand, were probably all septic, though in varying degree or nature. (They were not examined bacteriologically.) In two (Cases I and II) there is a clinical note that hypopyon was present at some stage, and in these cases there was also a small amount of pus in the vitreous; but in all three there were evidences of contamination, though the virulence of the infection must have been slight in all, as we are only too familiar with the extremely virulent cases in which acute purulent panophthalmitis rapidly supervenes, and all tendencies towards repair are rendered abortive.

The second fundamental point to be held in view is the period which has elapsed between the infliction of the wound and the examination. Thus, in the experiments we have:—V, 14 days: I, 15; III, 17; II and IV, 19; VI, 21; and in the cases:—I, 4 days; II, 8; III, 19.

The third point is the relative severity of the wounds in the various instances; and here many factors have to be taken into account. Of prime importance is the implication of the choroid, or sclerotic, or both, in addition to the retina; for in no case are we here dealing specially with ordinary perforating wounds (except in the wounds of entry in the experiments), for even where all the coats of the eye were pierced, it was from within and not from without—a point of considerable importance. Further, I think that the injury of any considerable blood-vessel adds enormously to the severity of the wound. Speaking generally, however, they may be classified as follows. All the injuries implicated both the retinal and uveal coats, with the possible exception of Case III, in which the affection of the posterior part of the ciliary body may have been secondary. Experiment III was the slightest wound, and Experiment II was only a little more severe; then comes Experiment IV, in which an appreciable amount of haemorrhage occurred; then Experiments V,

and I and VI, in all of which the sclerotic was also injured, being perforated in the two latter, of which VI is made the most severe of all by the puncture of a fair-sized vessel. The cases are more difficult to classify according to this criterion, forgetting for the moment the far more essential element of sepsis. Here, I think, the site of the wound of entry plays a prominent part, those in which it is situated in the cornea, involving wound of the lens, leading to much more general disorganisation than when the perforation is purely scleral. From this point of view, then, we may regard Case III as of minimal severity; Case I, with a small, purely corneal wound, as of medium severity; and Case II, with a larger corneo-scleral wound, as of maximum severity. In the cases under consideration, the size, nature, and shape of the foreign body, its probable velocity, &c., were fairly uniform, and we may regard this factor as constant.

Turning now to the histological features in the healing of retinal, choroidal, and scleral wounds, we find that the process is, in its essentials, the same as in other parts of the body, but that in details it is modified by the specific characters of the tissues involved. As elsewhere there is invariably hæmorrhage followed by leucocytic infiltration (Stage I). This is succeeded by organisation or cicatrisation (Stage II). These stages must now be considered in the light of the data obtained, paying due regard to previous work upon the subject—both special (ophthalmic) and general.

(1) *Hæmorrhage.*—It is obvious that even the slightest wound of the mammalian retina must be accompanied by hæmorrhage, since the non-vascular part of the retina lies between two vascular layers. In the peripheral parts the hæmorrhage may be minimal and little more than capillary. Here, mere endothelial walls are ruptured, and they probably play little part in subsequent events. In all cases, however, red corpuscles and leucocytes are set free, together with the plasma which provides some, at any rate, of the precursors of fibrin. In every instance, therefore, the wound is rapidly

filled with a clot, consisting of fibrin, with red corpuscles and leucocytes in its interstices. Now, the hyaloid membrane of the vitreous and the so-called internal limiting membrane of the retina have also been wounded for a corresponding extent, and the blood is thus given access to the vitreous. If a larger vessel has been wounded blood is poured out in the direction of least resistance, *i.e.*, along the track of the knife or f.b. The intra-ocular tension has been lowered, and in some cases nullified, but the consistence of the vitreous offers resistance to an equal distribution of the exudates. The retinal, and probably the choroidal, arteries exist normally in ideal anatomical surroundings for rapid retraction, whereby their elastic coats are able to bring their whole force to bear upon the blood-stream, and thus the outflow is minimised. Where this condition is absent, as in arterio-sclerosis, &c., hæmorrhage is followed by much more serious consequences (unless, indeed, it occurs in the otherwise intact eye, where the normal raised intra-ocular tension affords a similar support).

There is no evidence that the red corpuscles and fibrin play any active part whatever in the subsequent events. We, therefore, proceed at once to consider the leucocytes.

(2) *Leucocytosis*—(*a*) *The Origin of the Leucocytes.*—We have seen that Tepljaschin agrees with a minority of other pathologists in regarding some of the leucocytes as derived from the wandering cells of the vitreous. For my own part I have failed to find the vitreous play any but a purely passive part in any pathological condition, so that I have come to regard it as inert, and to look upon such terms as "shrinking of the vitreous," &c., as implying an activity which does not exist. At the same time, only the most painstaking investigation could really prove this to be the case, and the undoubtedly great *relative* inertness of the vitreous elements is sufficient excuse for leaving this point in abeyance.

There can be no doubt that the enormous majority, if not all, of the leucocytes are derived from the vascular (both

blood and lymph) channels. In some animals, the perivascular lymph-sheaths of the retinal vessels are conspicuous, and it is probable that their contained lymph affords its quantum of leucocytes.

As to the question whether all the leucocytes are the result of emigration, or whether those which have already emigrated, divide and multiply, it is probable that the latter view is correct, but my observations give no evidence. It may be remarked that the eye is admirably adapted for the solution of this and kindred questions, all that is required being the examination of earlier specimens by specific methods directed to the particular point.

(*b*) *The Function of the Leucocytes.*—It can scarcely be doubted that this is exactly the same as elsewhere in the body in similar circumstances. (*a*) *Digestive.*—By their ferment action they soften and liquefy the necrosed tissues, *i.e.*, those which have been killed by the severity of the injury. The products of digestion, now in solution, are readily carried off by the lymph stream. (*β*) *Mechanical.*—By their aggregation and fibrin-forming process they form plugs which help to seal up the broken and contused blood- and lymph-channels. This function has also a deleterious action in impeding the flow of nourishment to the part, thus increasing the necrosis, which they further deal with by their digestive function. It also impedes the outflow of effete material, thus leading to unfavourable conditions of vitality, which also must be taken into account. (*γ*) *Phagocytic.*—It is unnecessary to dilate upon the bactericidal function of leucocytes here, but their phagocytic activity is most easily seen in the very marked way in which they deal with the red blood corpuscles and with the blood pigment of broken down corpuscles (Fig. 2). This function is doubtless applied to all the more insoluble products of disintegration.

Whether the digestive activity of leucocytes is exerted upon the fibrin which they have helped to form, is doubtful. One is familiar with old-standing fibrinous clots containing multitudes of leucocytes, where there is no reason to suppose

that such a process has occurred, and it is probable that the fibrin is too admirably adapted for fodder for the young connective-tissue cells for it to be wasted in this manner.

(*c*) *The Fate of the Leucocytes.*—My experiments only afford evidence of one destination of the leucocytes, viz., disintegration. This is frequently observed in the broken-down white cells, with fragmented nuclei, and in the occurrence of free nuclei.

It can hardly be supposed that this is the fate of all of them, but I have seen no reason to suppose that they contribute directly to the formation of fibrous tissue. We have seen that the relative proportions of leucocytes to embryonic connective-tissue cells varies enormously at different periods and under different circumstances, especially sepsis, injury of the larger retinal vessels, wound of the choroid, &c. However few or however numerous they may be, they are always absolutely characteristic, and one never finds forms intermediate between them and connective-tissue cells. This I know is diametrically opposed to Tepljaschin's views, but it accords with the observations of Sherrington and Ballance, and of most other pathologists. It is another point which demands reinvestigation by special methods on early specimens.

Mention has been made of the extreme variability in the number of leucocytes found in different stages after injury in different conditions. This is a very striking point, and the most important factor seems to be the bacterial one. None of the cases described were severe cases of septic wound, but one is familiar with the rapid onset of purulent panophthalmitis in many such. Here, the efforts towards repair, which we cannot doubt are exerted, are entirely abortive, and Stage II is never reached. The variability in number, however, is very noticeable in the aseptic cases, and seems to be largely dependent upon the severity of the wound, *i.e.*, upon the injury of large retinal blood-vessels, or of the choroid, or upon actual perforation. Probably the implication of the choroid is the most important factor, and this gives some

support to the hæmic origin of the cells. The more purely retinal wounds with a minimum of bleeding have very few leucocytes, but wherever there is much hæmorrhage the white cells are abundant.

We have now considered the first stage in so far as it affects the actual site of the wound. The changes in the surrounding tissues are very little marked in aseptic wounds, being limited to the ordinary signs of acute inflammation in the immediate vicinity. In septic wounds the inflammatory process is much more widely spread, resulting in congestion (most marked in the choroid), and infiltration with round cells. These changes, however, are amongst the earlier ones of which the experiments and cases offer no good example.

In the second stage, the essential part of the process of healing commences and soon gains the upper hand, consisting of the formation of fibrous tissue, leading to the production of an organised cicatrix.

(3.) *Cicatrisation.*—(*a*) *The Formation of Fibrous Tissue.*—As stated above, we have seen reason to deny the formation of fibrous tissue from leucocytes. We must, therefore, look to the fixed tissue elements for the mother-cells. We are at once confronted with the number of tissues to choose from. Putting the vitreous out of the question, as before, we have in all the specimens the various structures of the retina and choroid to deal with. The true neural elements of the retina for the most part degenerate, the ganglion cells and their nerve-fibres never regenerate, and even if the rods and cones make abortive attempts (Tepljaschin, &c.), these are merely curiosities, and form no essential part of the cicatrix. We are thus reduced to the neuroglial elements of the retina, its blood-vessels, and the choroid.

Proliferation of Müller's fibres has been described in innumerable pathological cases in human eyes; exactly how much credence is to be attached to these cases is doubtful. The normal condition of Müller's fibres is very difficult to determine, except in isolated teased specimens. They are so overwhelmed by the nerve cells in the normal state that

only parts of them can be seen peeping out here and there. In the pathological cases, the nerve elements are invariably more or less degenerated—and then Müller's fibres are said to be hypertrophied and to have proliferated. Confining ourselves only to the specimens reported in this paper, I have failed to find any evidence whatever of proliferation of Müller's fibres. The question cannot be said to be finally settled until it is investigated with this special aim in view, using specific neuroglial stains and examining carefully at various stages. It may, indeed, be found necessary to apply such tests to wounds in very young animals. In ordinary aseptic wounds I have not observed such direct continuity between Müller's fibres and the new fibrous tissue such as is depicted in Tepljaschin's paper, Plate xxii, Fig. 4; whereas his Fig. 28, Plate xxiii, well represents the appearances found in Experiment II, Figs. 6 and 7, and resembles those found in Experiments III and IV, Figs. 8 and 12. Indeed, the differentiation between the two tissues is usually very distinct even by the ordinary methods of staining, but an apparent continuity is not infrequent in pathological specimens. Differential staining can alone determine how far the appearances are deceptive. It is perhaps *à priori* improbable that epiblastic elements should take any very prominent part in a process so essentially characteristic of mesoblastic tissues. Chemical tests may also be valuable in elucidating this point, and it would be interesting to know the relative part played by epi- and meso-blastic structures respectively in other cases of sclerosis of the central nervous system, for, as far as I know, the question has received scant attention by neurologists. It would probably be easier to decide in these cases, and the analogy is so exact that the result would almost certainly be applicable to the retina.

We are reduced, therefore, to the mesoblastic tissues found in and around the retinal blood-vessels and in the choroid. There is ample evidence to show that both of these structures take part in the process, though the latter is by far the more important, chiefly by virtue of mere mass.

The slighter the injury to the choroid, the less is the hyperplasia of connective-tissue, and, though no example can be adduced from the specimens examined, I have little doubt that a pure retinal lesion becomes cicatrised wholly from elements derived from its own mesoblastic elements, *i.e.*, from the connective tissues in and around its vessels. Where the fibrous tissue is seen growing over the normal or degenerated retina (*e.g.*, in Experiment IV in the posterior part (see description) and in Fig. 12), the choroid being intact or not in direct continuity, it is doubtless derived from the retinal vessels alone.

The persistence of the retinal blood-vessels is a striking feature even in the septic cases. When all other parts of the retina have melted away, and are replaced by masses of leucocytes (*e.g.*, Fig. 17), these stand out clearly—widely dilated, often crammed with red corpuscles, or partially or completely thrombosed. The appearances seen under high magnification in these and cognate cases, lead one irresistibly to the conclusion that the cells of the adventitia proliferate, and add to the mass of embryonic tissue. This is seen in an early and feeble phase in Fig. 17, and in a later and strenuous phase in Fig. 18.

Where the wound results in injury to the sclerotic, no new factor is introduced, at any rate in the early stages, unless this coat is perforated, and then the episclera assists in the process in exactly the same manner as the choroid, only in less degree, due doubtless to its inferior vascularity. In anterior perforating wounds, the episclera takes a larger share on account of its relatively greater vascularity here, due to its more intimate association with the extrinsic muscles. Only the fibrous tissue elements in the muscles proliferate, the muscle fibres remaining inert.

The origin of connective tissue (and other cells) from the cubical cells of the pars ciliaris retinæ has been much insisted upon from time to time. Appearances indicating this are not wanting in my specimens, but I think they are fallacious. The cubical cells are much altered in appearance

in those cases in which new filaments of fibrous tissue stretch forward through the vitreous as far as the ciliary body. Some of them are stunted, whilst others, often forming groups, are much elongated, so that they become cylindrical. The inner ends of these elongated cells are always attached to the fibrous filaments, and their elongation is probably due to traction. By the time the filaments have reached so far forwards, the posterior mass of new tissue is commencing to shrink—a characteristic common to all cicatrices—and thereby considerable traction is exerted upon the anterior attached ends. The sweeping curves of the filaments are probably conditioned by the effect of the track of the wound in the vitreous upon its normal structure, and results in the ciliary retinal cells being more often drawn forwards and inwards than directly inwards or inwards and backwards.

Having stated so much, it still remains to be shown which of the mesoblastic tissue elements provides the actual mother-cells of the new formation. The specimens provide little evidence for proving this point, one of the most difficult in pathology. Attention may be directed especially to the work of Sherrington and Ballance upon the subject (*vide supra*, p. 219).

(*b*) *The Behaviour of the Endothelial Cells.*—Endothelial cells are found normally only in the blood and lymph vessels of the retina and choroid, with the possible addition of an incomplete layer lining the hyaloid membrane, and a layer in the choroid which is probably a vestige of the tapetum. It is certain that they proliferate during the repair of wounds and in other pathological conditions. They are most commonly met with in various inflammatory conditions, as a uni- or multi-cellular layer covering the inner surface of the retina. In such cases they may well be derived from the cells lining the hyaloid, but this is purely hypothetical. They seem to vary greatly in numbers and distribution in different wounds, and it is difficult to assign the reasons for this peculiarity. In Experiment I they were present in large numbers in the vitreous, especially in a band,

of which Fig. 3 represents a part; but they are also generally present amongst the embryonic connective tissue, particularly in the early stages. They are not infrequently present also in later stages, and in very chronic inflammatory conditions. I have not seen such evenly arranged layers over the retina in the case of wounds as in other pathological states, but they are recognisable amongst the fibrous tissue in Figs. 12 and 14.

It has been stated that the endothelial cells become changed into new fibrous tissue, and that this is their main function in these cases. I have not seen any evidences of transition states, the two types of cells being usually quite characteristic. I believe it is not yet definitely settled which layer of the embryo they originate in, so that their developement does not assist us in determining their probable destination. It is probable that their function in pathological conditions has not, in the past, received the attention it merits.

Remarks on Retinitis Proliferans.

It is unnecessary to enter into details as to the causation and general pathology of Retinitis Proliferans in this place. An excellent *résumé*, with the best recorded pathological examination of a case, will be found in a paper by Mr. Percy Flemming in the Transactions of the Ophthalmological Society of the United Kingdom.[25] The crucial point in the pathology of these cases is the explanation of the widespread proliferation and organisation which takes place, and which does not occur in other cases of retinal hæmorrhage. Mr. Flemming attributes this to the "inflammatory process present at the time in the retina and papilla," due to some toxic agent (*e.g.*, that of diabetes, syphilis, or nephritis). I think the sequence is probably as follows. Some toxic condition leads to a retinal hæmorrhage. If it is situated in the peripheral parts of the retina, or is due to rupture of the smaller vessels nearer the papilla—in other words, if it is a

small hæmorrhage—no large proliferation of new tissue will result. A scar will result from the organisation of the clot through the medium of the scanty mesoblastic tissue of the walls of the smaller retinal vessels. If, however, a large hæmorrhage occurs, it will be in the neighbourhood of the disc, or if caused by repeated hæmorrhages in slightly more peripheral parts, will yet invade the neighbourhood of the larger vessels, *i.e.*, again the neighbourhood of the disc. Now it is around the larger vessels, and *par excellence* upon the disc that the main mass of retinal mesoblastic tissue is situated. At the papilla itself, we have not only the walls of the vessels, but also remnants of the hyaloid artery and a ring of anastomoses with posterior ciliary vessels—in fact, the largest mass of mesoblastic tissue found in the walls of the true optic cup. The nearer the hæmorrhage approaches this point, and the more widespread its traumatic and irritating effects in this immediate neighbourhood, the more tissue will be excited to reparative reaction, and the greater will be the proliferation and organisation. Hence the fact that Retinitis Proliferans invariably springs from the disc or its vicinity. In the periphery there are two factors acting against such proliferation : first, the smallness of the hæmorrhage, which, however, may possibly be increased by repetition or multiplicity and confluence; second, and more important, the small amount of tissue which is capable of proliferating and inducing organisation.

Upon this hypothesis, the toxic agent acts only, or at any rate chiefly, by producing retinal hæmorrhage, and only secondarily, or not at all, by producing an inflammatory process in the retina and papilla. Against the latter hypothesis are the facts that the relatively healthy retina may show no marked signs of any such inflammatory process being present, and that the toxic conditions so frequently present, whilst potent causes of hæmorrhage and even of inflammatory conditions, are, with the possible exception of nephritis, not known to be specially effective in leading to fibrous tissue proliferation.

The experiments described in this paper were performed in the Physiological laboratory, University College, London. I desire to express my thanks to Professor Starling for the use of the laboratory. The expenses were defrayed by a Royal Society grant. The histology was worked out in the Pathological Laboratory, R.L.O.H.

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A CASE OF RETINITIS PIGMENTOSA, WITH PATHOLOGICAL REPORT.

By W. T. Lister, F.R.C.S.

I am enabled to bring forward this case of Retinitis Pigmentosa owing to the kindness of Mr. Nettleship, who both gave me the specimens and also supplied me with his notes of the case made 24 years ago. We are both greatly indebted to Dr. F. Pritchard Davies for having sent the specimens to Mr. Nettleship, after the case had been under his care as an inmate of the Kent County Asylum for 20 years.

July, 1878. W. S., male, æt. 38, came to St. Thomas's Hospital complaining that he had always been "near sighted," and had never been able to see in the dusk as well as other people.

His right eye had begun to get worse in 1870; he had been told he had cataract in the eye; and an operation for artificial pupil had been performed downwards. His left eye began to fail in 1876.

R.V. J. 19; c. sp. – 8 D 20/100.

L.V. J. 1 with difficulty at 4 inches; c. sp. – 6 D. 20/70.

Media.—Posterior polar and cortical opacity in each lens—denser in R. than L.

Fundi.—Advanced Retinitis Pigmentosa, with much pigment. The pigmentation was situated in a somewhat zonular area, extending in thinly scattered spots as far as the disc, but ceased, to a great extent, towards the periphery. The optic discs were pale; the vessels were small; there was a myopic crescent and some diffuse myopic changes of the adjacent choroid, but no islands of atrophy of the choroid.

W. S. had had good health; there was no history of syphilis.

Family History.—His mother died insane, æt. 72. He had one brother who could not see in the dusk; one half-brother and

one half-sister, both mentally defective. No consanguinity of his ancestors was known.

It was learnt from his wife, in 1880, that W. S. was suffering from delusions, and, later in the year, Dr. F. Pritchard Davies, Superintendent of the Kent County Asylum, wrote to tell Mr. Nettleship that W. S. was under his care with religious melancholia.

In 1900, Dr. Davies again wrote to Mr. Nettleship, and stated that W. S., who for some years had been totally blind, had just died, æt. 60, and he kindly sent both globes for examination.

Pathological Examination.—Both globes had been preserved in spirit, which had made the tissues, and especially the retina, very brittle. Nothing abnormal was noted before freezing and opening the eyes by an antero-posterior section in one eye and an equatorial section in the other.

The anterior chamber was shallow; the iris normal; the lens opaque throughout; the vitreous was fluid. Retina and choroid *in situ*, but so intimately united that separation of the two was impossible.

The retina in its posterior two-thirds was deeply pigmented, the pigmentation taking the form of an irregular network, which gave it a speckled appearance. (Fig. 1, *c*.)

The anterior third of the retina (Fig. 1, *b*), forming a zone about 7 mm. in width, was pale and unpigmented, strikingly different, and sharply marked off from the posterior pigmented portion. (Fig. 1, *c*.)

The macular region appeared as an oval pale grey area; the pigmentation here also was much less than in the surrounding parts. There was a well-marked myopic crescent on the outer side of each disc.

The *retinal vessels* were extremely minute, but with a magnifying glass the main trunks could easily be made out as white lines running over the retina, and, in places, a very fine dark line down the centre of the pale path could be distinguished.

The retina and choroid, as mentioned above, were intimately adherent; together they could be separated from the sclerotic with difficulty, though in many places they were very adherent.

Portions of the combined retina and choroid were mounted "on the flat" (Figs. 1 and 2), to show the distribution of the pigment and its relation to the blood-vessels, and microscopic sections were made of the—

(1.) Lens.
(2.) Optic nerve and disc (longitudinal).
(3.) Retina and choroid.

Sections of the lens show the ordinary cataractous changes; nothing distinctive could be made out at the posterior pole.

Sections of the optic nerve and disc show the nerve to be more fibrous than usual, the septa being thicker than normal and particularly well marked. (The specimen having been preserved in spirit, no special stains were employed to show degeneration of the nerve fibres.)

The optic disc is slightly cupped and is covered by laminated connective tissue, which is continued into the surrounding retina, and takes the place of the nerve-fibre layer.

Sections through the retina show appearances similar, on the whole, to those described by previous observers.

Only in the parts close to the disc and macula is there any trace of the normal retinal layers. In the darkly-pigmented area the retinal tissue is completely atrophied, and its place is taken by a loose connective tissue.

Taking, first, the sections of the parts close to the disc and macula:—

The *nerve-fibre layer* shows no thickening, nor is there any adventitious layer formed in the place of the membrana limitans interna, as in the specimens described by Leber and Landolt. The nerve fibres have been replaced by loose laminated connective tissue, as mentioned above, which is continuous with that found on the surface of the disc.

The *ganglion layer* is well seen in the macular region, but the cells are not so closely packed as normally.

The *inner reticular layer* can be distinguished in places near the disc and macula.

There is only one *nuclear layer* visible, and this is much thinner than either of the two layers that are usually found. (As there are not two nuclear layers in any part, it is impossible to say for certain whether this remaining layer represents the outer or the inner nuclear layer or both; but from its special relation to the inner reticular layer and the ganglion layer, it probably represents the inner nuclear layer.)

The *layer of rods and cones* is altogether absent. Its place has been taken by laminated fibrous tissue, between the layers of which are seen numerous oval nuclei (Figs. 3 and 4). This tissue lies next to the pigment epithelium on the outer side and to the nuclear layer on the inner side.

The pigment epithelium is a well marked layer in the macular region.

In the sections through the retina outside the central area practically all traces of the normal structure of the retina are lost. Here and there a short line of pigment cells at the site of the pigment epithelial layer is seen, and again somewhat linear groups of nuclei resembling those in the nuclear layers are found, but as a whole the tissue representing the retina consists of connective tissue, which is loose and reticular towards its inner aspect, and laminated and much more dense towards its outer aspect (Figs. 3 and 4).

In this connective tissue are embedded the blood-vessels. These have thickened and deeply-pigmented walls, and in most the lumen is blocked (Figs. 3 and 4). Some of the vessel walls have retained their laminated structure (V_2), but others have undergone hyaline degeneration, and are structureless (V_1).

There are a few irregular excrescences from the choroid in the outer part of the connective tissue; these also are pigmented, and probably have been derived from the lamina vitria, but this layer cannot be made out.

On examining the portions of retina and choroid mounted on the flat (Fig. 2), the network of pigment is clearly seen; and when viewed under the microscope it is evidently on a

different level from the choroidal pigment. One sees at once also that the pigmentation follows to a great extent the course of vessels, but these vessels are neither the main retinal trunks, nor are they capillaries, but are probably small arterioles from which the capillaries would be given off. I think it is quite possible that some of these vessels are of new formation.

Here and there more diffuse patches of pigment are seen, and some of these are probably the pigmented excrescences from the lamina vitria.

The choroid is atrophied, and the chorio-capillaris layer has disappeared. But it is a striking fact that the choroidal vessels seen in the sections have not got thickened walls. This corresponds with the appearance of the choroidal vessels seen on the flat, which have no irregular vermicular course as is so commonly seen in cases of sclerosis of the choroidal vessels.

Beyond the description of this case I have little to contribute to the pathology of the disease. The changes found in cases of retinitis pigmentosa, taken as a whole, are concentric atrophy of the retina with pigmentation and degeneration of the retinal vessels, associated with a variable amount of chronic inflammatory change.

Some hold that these changes are brought about by primary vascular disease either of the retina or choroid. But the concentric defect corresponds with a *nerve distribution* rather than a *vascular* distribution, and therefore I think we must look for a degeneration commencing primarily in the nervous tissues rather than in either vascular system.

There is undoubtedly a group of cases with sclerosis of the choroidal vessels and—probably a secondary—pigmentary degeneration of the retina; but these cases must be classed separately, for they are often unsymmetrical or even entirely monocular, whereas typical cases of retinitis pigmentosa are symmetrical, and no changes are seen in the choroid or its vessels, which, if present, would be especially apparent owing to the atrophy of the retinal pigment.

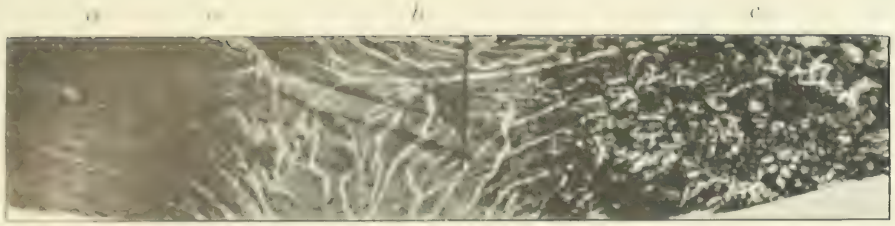


Fig. 1.

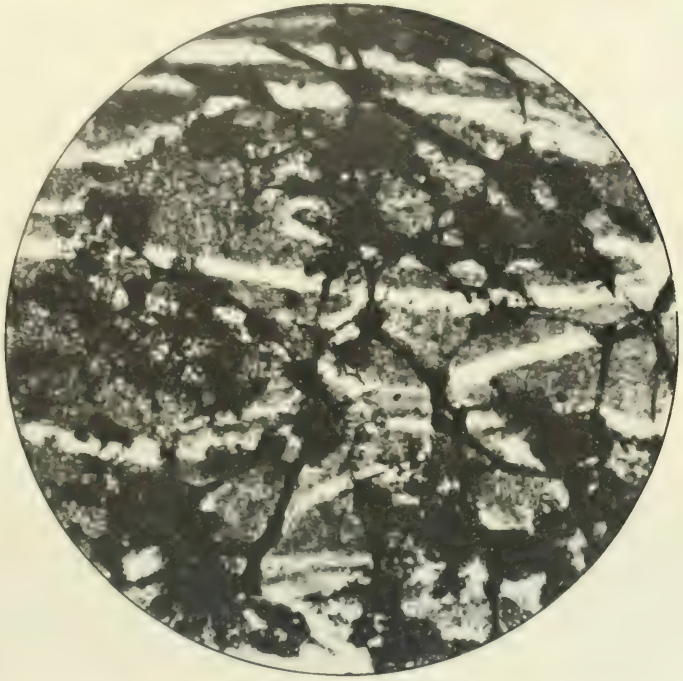


Fig. 2.

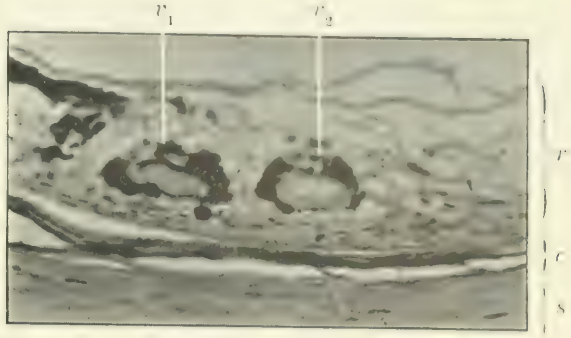


Fig. 3.

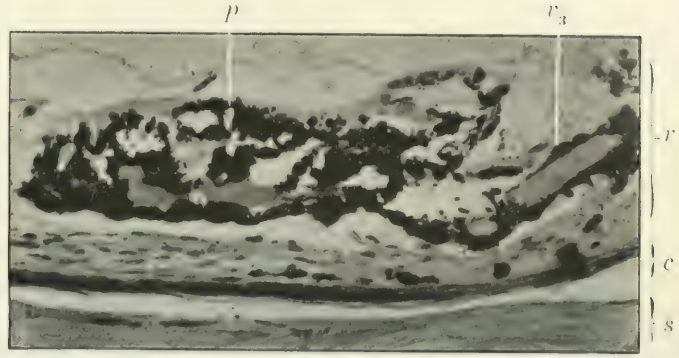


Fig. 4.

FIG. 1.—Strip of retina and choroid mounted on the flat. (*a*), ciliary body; (*o*), ora serrata; (*b*), unpigmented zone of the retina, showing the choroidal pigment and the course of the choroidal vessels; (*c*), pigmented portion of the retina.

FIG. 2.—Pigmented portion of retina with choroid, mounted on the flat, under higher magnification. The black pigment in the retina is easily distinguished from the paler choroidal pigment. The black retinal pigment is arranged (1) in lines, following the course of blood-vessels, and (2) in patches. The white lines between the areas of choroidal pigment mark the course of the choroidal vessels.

FIG. 3.—Section through retina (*r*), choroid (*c*), and sclerotic (*s*). The retina is atrophied, its place being taken by connective tissue, which is loosely arranged internally and laminated externally; between the layers can be seen numerous oval nuclei.

Embedded in the connective tissue are seen the pigmented vessels in cross-section (v_1, v_2)), in both of which the lumen is blocked, but the walls of (v_2) still retain their laminated structure.

FIG. 4.—Also taken through retina, choroid, and sclerotic; shows in the main the same points as Fig. 3.

In addition (v_3) is a pigmented vessel cut longitudinally, and at (*p*) the section passes through one of the larger pigmented patches seen in Fig. 2.

THE OPHTHALMIC ARTERIES IN THE RABBIT AND DOG.

By E. E. Henderson, F.R.C.S.

In any experiments on the intraocular circulation one of the lower mammals must, as a rule, be employed. As the dog and rabbit have been the animals most employed for this purpose, it is of importance to the experimenter to ascertain the anatomy of the orbital arteries in these animals, and so note the differences from the human type. For this purpose six dogs and six rabbits have been injected with plaster of Paris, and careful dissections made on each side. As this injection only penetrates to the smaller arteries the more minute anatomy cannot be studied in this way. I propose, in this note, only to deal with the gross anatomy.

The only text-book devoted to the anatomy of the dog with which I am acquainted is that of Ellenberger and Baum.[1] In dealing with the point now under discussion these authors refer to the work of Bellarminow, but do not give the reference, and I have been unable to consult the original paper. The standard authority for the anatomy of the rabbit is Krause.[2]

As is well known the blood supply of the eye as we descend the animal scale tends to be derived more and more from the external carotid. It is therefore only to be expected that individual variations would be of frequent occurrence. I will first briefly describe the results of my dissections, and compare them with the account of the blood supply of the eye, as given by the authors referred to above. The rabbit, as being apparently of a somewhat more standard type, will be taken first.

The common carotid in the rabbit gives off the thyroid artery and the ascending pharyngeal. In addition, Krause adds that the laryngeal may come from it. At the level of the hypoglossal nerve the artery divides into the internal

and external carotids. The former is much the smaller, and runs without branching to the bulla tympani, and enters the skull by the carotid foramen. It then winds round the lateral side of the body of the sphenoid bone, and after crossing the oculo-motor nerve, gives off the anterior communicating and superior ophthalmic arteries. It ends by dividing into the anterior and middle cerebral. In four of my dissections I have been able to trace a fine branch of communication between the internal carotid, as it lies on the side of the body of the sphenoid, and the internal maxillary close to the origin of the inferior ophthalmic artery. The external carotid gives from its inner side the laryngeal, lingual, and external maxillary in that order from below upwards. The last-named artery gives off the facial. From the posterior wall is given off the occipital. A little below the neck of the jaw the artery divides into the superficial temporal and the internal maxillary. The former gives off the transverse facial and the auricular. The internal maxillary runs deep to the internal pterygoid behind the lower jaw. It gives off the tympanic, the inferior alveolar, some muscular branches, and the middle meningeal. It then passes through the pterygoid canal, and gives off the inferior ophthalmic. It ends by supplying a superior alveolar branch, and divides into the infraorbital and pterygo-palatine arteries.

So far in my dissections the arrangement of the branches of the carotid arteries are in entire agreement with the description given in Krause (*loc. sup. cit.*, p. 183). I have, therefore, followed his nomenclature. I now quote in full his description of the ophthalmic arteries.

"The inferior ophthalmic artery runs on the anterior surface of the upper part of the great wing of the sphenoid, bends forward over the optic nerve, and reaches its anterior side. It there anastomoses with the superior ophthalmic artery. Its branches are as follows:—1. Lacrymal, which appears through the posterior supraorbital foramen as the supraorbital artery. 2. Frontal, which in the same way goes

through the anterior supraorbital foramen; immediately after its origin it usually gives off the well-developed anterior ethmoidal artery, which goes through the ethmoidal foramen into the nasal cavity. 3. Muscular branches to the eye muscles.

"The superior ophthalmic artery is but little developed. It runs forward to the optic foramen, and goes through this on the under and lateral side of the optic nerve into the orbit, winds under the nerve to its anterior side, and anastomoses with the inferior ophthalmic artery. It gives off the ciliary arteries and the central artery of the retina." (*Loc. sup. cit.*, pp. 185, 186.)

In two of the dissections I have been unable to trace any anastomosis between the two ophthalmic arteries in the situation here described. In both these cases the anastomotic branch between the internal carotid and the internal maxillary was present. In two others this anastomotic branch was also present, and in these the superior ophthalmic artery was very small.

In the dog the carotid artery gives off as its first branch a thyroid artery. This in all the dissections has been the superior, but Ellenberger and Baum say that occasionally the inferior thyroid may also arise from the common carotid immediately after its origin. A muscular branch to the sternomastoid usually arises at about the same level as the thyroid. A little higher the ascending pharyngeal and the laryngeal arise. The artery now divides into the internal and external carotids. The internal carotid is always the first postero-external branch. It takes origin just at the level of the hypoglossal nerve. It is most easily reached in the living animal by retracting the common carotid outwards and tracing up its inner side. There is always present an enlargement close to its origin, which may possibly represent a carotid gland. The artery runs straight up with the vagus to the bulla, and enters the skull through the carotid foramen. It gives no branches in the neck. The external corotid gives as its first branch on the outer side the occipital. This artery

arises close to the internal carotid, and is of about the same size. In the living animal it may readily be mistaken for it. It is somewhat more superficial in origin, and will usually be found to give an anterior branch soon after its origin. The next branch and one of the largest is the lingual. Then comes the external maxillary, from which is derived the facial. The artery then breaks up into the superficial temporal, auricular, and internal maxillary arteries. The internal maxillary winds round the maxillary joint over the bulla, enters the pterygoid canal, and ends after leaving this by dividing into the infraorbital and palatine branches. Its branches may be divided into two sets, those given off before it enters the pterygoid canal, and those given off after it leaves it. The first set comprise the middle meningeal, the deep temporals, the inferior alveolar, and the muscular. The second set comprise a further set of muscular branches and the inferior ophthalmic.

Up to the point of origin of the ophthalmic arteries the dissections have corresponded with the description of Ellenberger and Baum, whose nomenclature I have followed. As some differences of detail have been noticed in the origin and communications of the ophthalmic arteries, it seems advisable to give their account in greater detail.

"The ophthalmic artery springs from the internal maxillary after it has left the pterygoid canal, and next runs between the periorbita and the temporal muscle, and further in on the orbital part of the frontal bone on the outer side of the periorbita to the ethmoidal foramen. Through this it runs into the skull as the ethmoidal artery. Shortly after its origin it, or one of its muscular branches, gives an anastomotic branch, which goes to the internal carotid. This, according to Bellarminow, who calls it the internal ophthalmic artery, gives off the central artery of the retina. The ophthalmic artery has the following branches :—

"1. Two muscular branches for the eye muscles, from one of which the rectus lateralis and superior, the periorbita orbital fat and tear glands are chiefly supplied ; from the

other the rectus medialis and inferior and the obliquus lateralis. Both often arise from a common stem. They anastomose with the frontal artery and the malar branch. From them small branches run to the bulb and perforate the sclera as the anterior ciliary arteries.

"2. The lacrymal artery, which accompanied by the nerve of the same name runs on the rectus lateralis, and divides into branches to the lacrymal gland and upper lid.

"3. The frontal artery. This runs laterally on the superior rectus, and supplies this muscle, the rectus medialis, the obliquus lateralis, the orbital fat, and the periorbita, and reaches to the upper lid and lateral canthus, where it anastomoses with the superficial temporal.

"4. The posterior ciliary arteries—two arteries which accompany the optic nerve and by anastomotic branches form a network on it. At the entrance of the nerve into the bulb the two arteries form a ring on the sclera. From this ring on each side spring one long posterior ciliary and numerous short posterior ciliary arteries.

"5. The posterior ethmoidal artery. This runs into the skull through the posterior ethmoidal foramen, and then divides into two branches. These give off the anterior meningeal arteries, which anastomose with the arteries of the corpus callosum. They then leave the skull through the anterior ethmoidal foramen, and anastomose with the palatine and septal arteries." (*Loc. sup. cit.*, p. 186.)

In addition to this description there is given in small print, after the chapter dealing with the eye, the following account of Bellarminow's description of the ophthalmic arteries. "Bellarminow describes an external ophthalmic artery which rises from the internal maxillary artery and an internal ophthalmic artery from the internal carotid—(it is this which we have described as the ramus anastomoticus). The latter, much the smaller, gives off the central artery of the retina. The former, running nasalwards, divides into the medial iris artery, and a branch which, after reinforcement by one from the internal ophthalmic artery, becomes the

lateral iris artery. Forward in the choroid is formed an arterial ring, which is supplied by both iris arteries directly and indirectly through the mediation of the circulus iridis major, as well as by the choroidal arteries. From this branches are sent to the ciliary body and choroid."

In none of my dissections has this arrangement been exactly followed. In all of them the anastomotic branch from the internal carotid has entered the internal maxillary immediately before the origin of the inferior ophthalmic artery, and this latter has in all been represented by two separate branches. In consideration of the conditions found in the rabbit it is wiser to adhere to the terms superior and inferior, although Bellarminow's division into internal and external has the advantage of indicating their source from the internal and external carotids respectively. In only half the dissections has there been present a superior ophthalmic artery arising from the ramus anastomoticus. In the remaining half this artery has been derived from the internal carotid after the main trunk has left the internal carotid, and has entered the orbit through the optic foramen. The anastomotic branch is always of very fair size, and arising immediately after the internal carotid has reached the cavernous sinus, passes through the fissura orbitalis below the first division of the fifth nerve. In two dogs a large meningeal branch has arisen from the ramus anastomoticus on both sides. This branch has apparently taken in these cases the place of the middle meningeal.

If the anastomotic branch always supplied the interior of the eye it is certainly of sufficient size to maintain the circulation, and, as blood reaches it equally from the internal maxillary and internal carotid, it is probable that the supply to the eye would not suffer from the loss of only one of these sources. This, however, as has been shown, is not invariably the case. But in those cases in which the ciliary arteries and the central artery of the retina have been derived from a superior ophthalmic artery arising from the internal carotid, a free anastomosis has always been present between this

superior ophthalmic artery and one of the branches of the inferior ophthalmic artery. It is then, even in these cases, anatomically possible for the intraocular circulation to be maintained in the absence of one or other of the ophthalmic arteries.

In man a very different supply obtains. The anastomoses between branches of the ophthalmic artery and those of the external carotid are both few and far between. I will briefly enumerate them, taking as authority Quain's Text-book of Anatomy.[3]

The branches of the human ophthalmic artery with their anastomoses are there enumerated as follows (*loc. sup. cit.*, p. 408) :—

1. The central artery of the retina usually arises in common with the internal ciliary trunk, sometimes with the external. No anastomoses.

2. Ciliary arteries: A, posterior from ophthalmic; B, anterior from muscular and lacrymal. The former have no anastomoses, and the latter no direct one.

3. Lacrymal. This anastomoses with the anterior deep, temporal, and transverse facial, and through the outer end of the sphenoidal fissure with the middle meningeal.

4. Recurrent branch to anastomose with internal carotid.

5. Muscular branches. Some anastomoses between the infraorbital and these branches must take place in the substance of the inferior oblique, but are not directly mentioned.

6. Supraorbital. This anastomoses with superficial temporal.

7. Anterior and posterior ethmoidal. No anastomoses given.

8. Palpebrals. No anastomosis given.

9. Nasals. Anastomose with facial.

10. Frontals. No anastomosis given.

The following variations are also enumerated. The ophthalmic artery may enter the orbit through the sphenoidal fissure. The lacrymal artery not infrequently, and, in rare cases, a large part, or even the whole of the ophthalmic artery

itself, arises from the middle meningeal. The lacrymal artery may also be reinforced by the anterior deep temporal artery. The ophthalmic artery has been seen to give off the middle meningeal.

Fr. Meyer,[1] in discussing a case in which an abnormal course of the branches of the ophthalmic artery led to free arterial bleeding, and in which a subsequent post-mortem examination showed that the lacrymal and frontal arteries were derived by a common stem from the middle meningeal, sums up the literature of the subject and enumerates the possible varieties. There is, however, no mention made of the morphology. In view of the conditions found in the animals now under discussion, the occasional origin of the ophthalmic artery, or of some of its branches, from the middle meningeal, may be of morphological importance.

Note on the Venous Return of the Blood from the Eye in the Dog.

The blood from the interior of the eye is mainly carried off by the vorticose veins, a small quantity also escaping through the anterior ciliary veins and the central vein of the retina. The vorticose veins are generally four in number, which ultimately unite to form two main trunks. The inferior and external one joins the ophthalmo-cerebral or inferior ophthalmic vein. The superior and mesial trunk runs into the cerebro-facial or superior ophthalmic vein. The two ophthalmic veins communicate with each other in the back part of the orbit and run into the cavernous sinus. The inferior one usually also communicates with the tributaries of the internal maxillary vein. Both veins receive numerous tributaries from all the structures in the orbit. The superior vein at the inner canthus joins the nasal veins and becomes the superficial facial. The inferior vein runs under the external wall of the orbit, and at the anterior border of the masseter joins the superficial facial to form the common facial trunk. It receives muscular branches from

the temporal and masseter, and communicates with the internal maxillary. After the common facial has received the lingual it is known as the external maxillary, and this vein is joined by the internal maxillary to form the external jugular. Soon after its formation this vein sends a large communicating branch across the middle line to its fellow of the opposite side, and terminates by joining the internal jugular near the first rib. The blood from the cerebral sinuses is mainly carried off by three veins, the external and internal jugulars and the vertebral. Of these three the latter is said to be the more important. The two former vary reciprocally. The external being in most cases the largest. The internal jugular has a large communication with the vertebral vein immediately after its exit from the skull. A coarse injection, such as plaster of Paris, if injected into the external jugular vein, fails to reach the facial owing to the valves. There does not, however, appear to be anything to prevent it reaching the cerebral sinuses, and so the ophthalmic veins. If it is desired to make an injection of the facial veins, it is best to employ hot gelatine and inject through the carotid artery. The accompanying diagram will serve to show how extremely free the venous outflow from the eyeball really is.

References.

(1.) Ellenberger and Baum. Anatomie des Hundes. Berlin, 1891.
(2.) Krause. Die Anatomie des Kaninchens. Leipzig, 1868.
(3.) Quain. Text-book of Anatomy, vol. ii, pt. 2, p. 408.
(4.) Fr. Meyer. Zur Anatomie der Orbitalarterien. Morphologisches Jahrbuch, pt. 12, 1887.

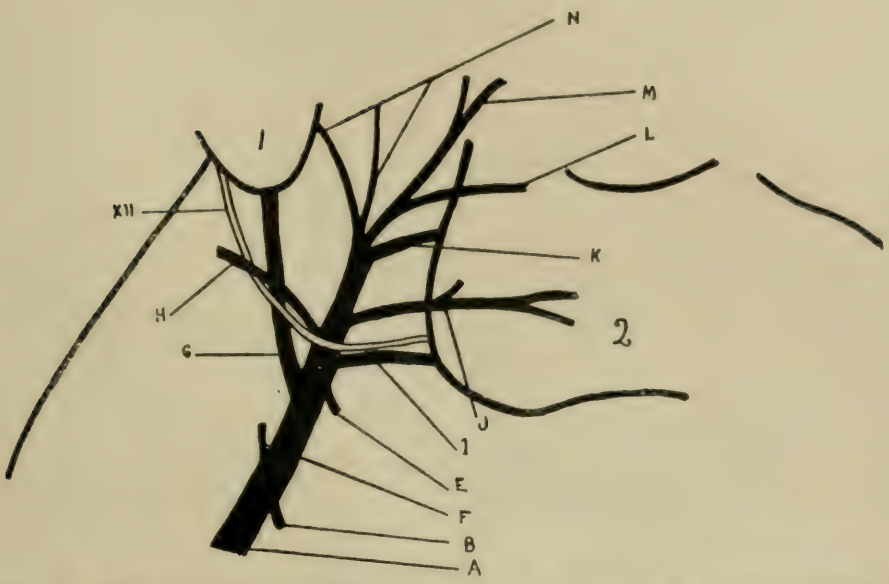


FIG. 1.—Scheme of the carotid arteries in the rabbit:—A, common carotid artery; B, thyroid artery; E, laryngeal artery; F, ascending pharyngeal artery; G, internal carotid artery; H, occipital artery; I, lingual artery; J, external maxillary artery; K, internal maxillary artery; L, transverse facial artery; M, superficial temporal artery; N, auricular artery. Hypoglossal nerve. 1, placed on the bulla tympani; 2, on the lower jaw.

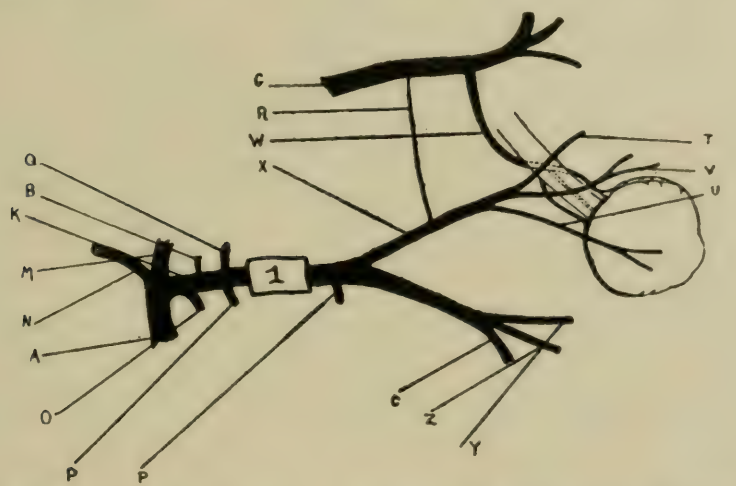


FIG. 2.—Scheme of the internal maxillary and ophthalmic arteries of the rabbit:—A, external carotid artery; B, tympanic artery; C, superior alveolar artery; G, internal carotid artery; K, internal maxillary artery; M, superficial temporal artery; N, auricular artery; O, inferior alveolar artery; P, muscular arteries; Q, middle meningeal artery; R, ramus anastomoticus; T, ethmoidal artery; U, lacrymal artery; V, frontal artery; W, superior ophthalmic artery; X, inferior ophthalmic artery; Y, infraorbital artery; Z, pterygopalatine artery. 1, placed on the pterygoid canal.

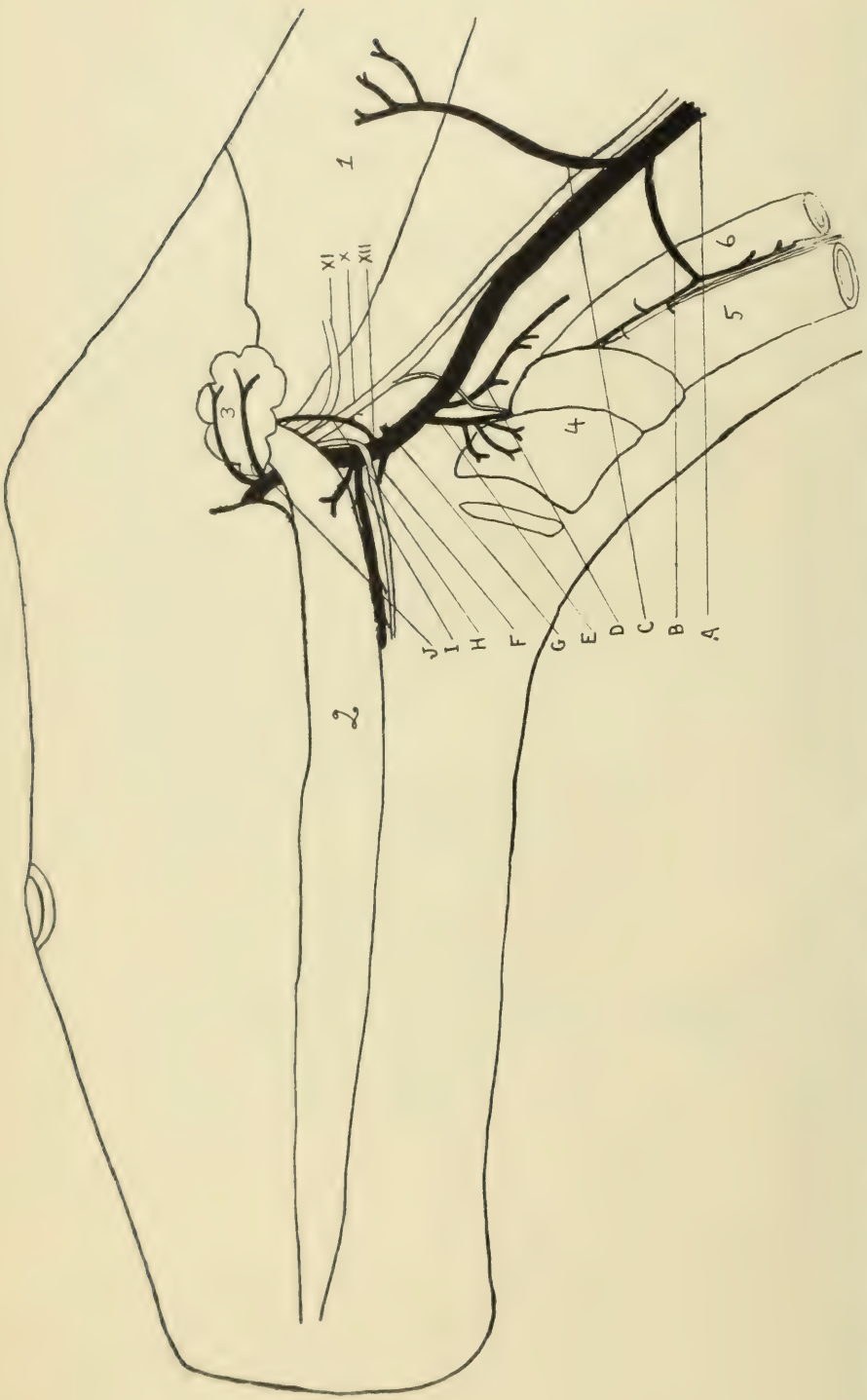


FIG. 3.—Scheme of the carotid arteries in the dog:—A, common carotid artery; B, thyoid artery; C, muscular branch to sternomastoid; D, muscular branch to deep neck muscles; E, laryngeal artery; F, ascending pharyngeal artery; G, internal carotid artery; H, occipital artery; I, lingual artery; J, external maxillary artery; X, XI, XII, vagus, spinal accessory, and hypoglossal nerves. 1, placed on the sterno-mastoid; 2, on the digastric; 3, on the salivary gland; 4, on the larynx; 5, on the trachea; 6, on the œsophagus.

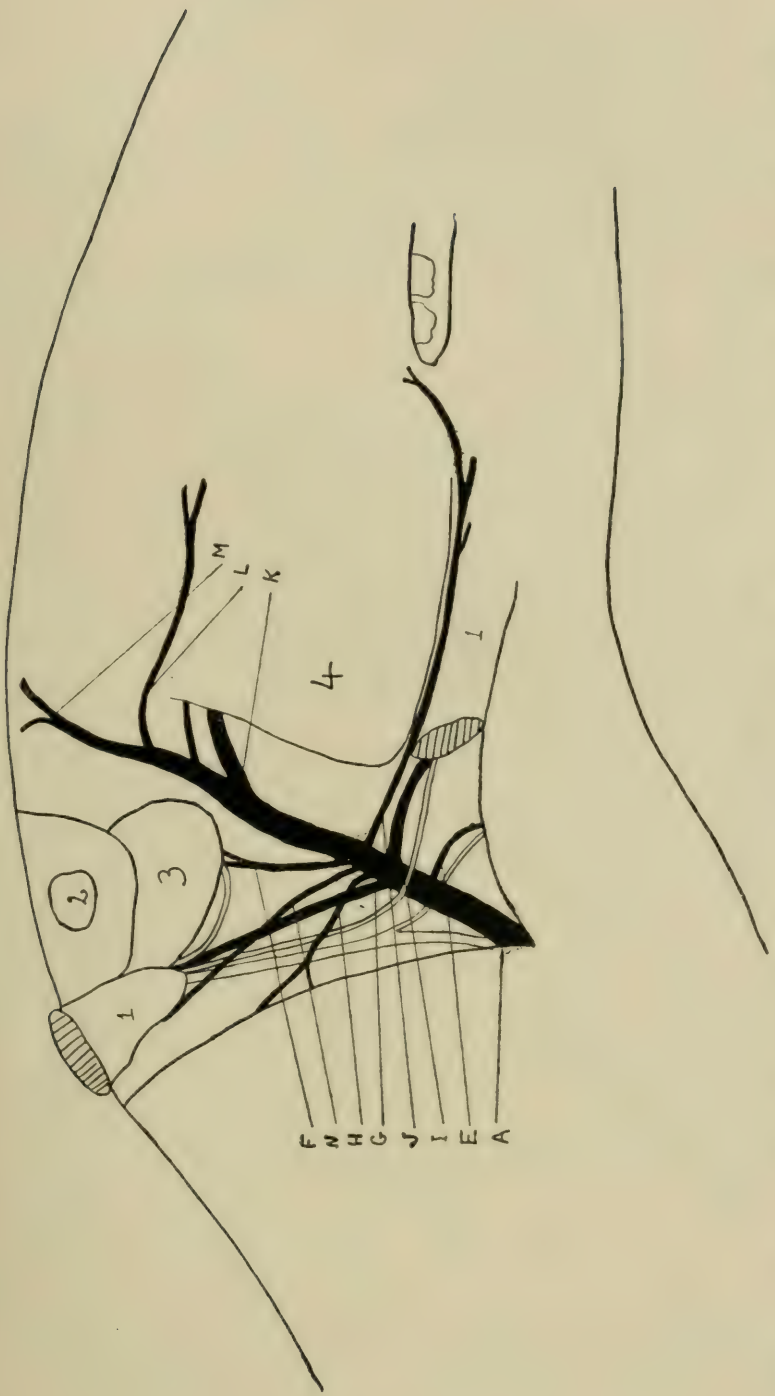


FIG. 4.—Scheme of carotid arteries in the dog, traced higher after division of the digastric:—A to N as in Fig. 3. 1, placed on the digastric; 2, on the ear; 3, on the bulla tympani; 4, on the lower jaw.

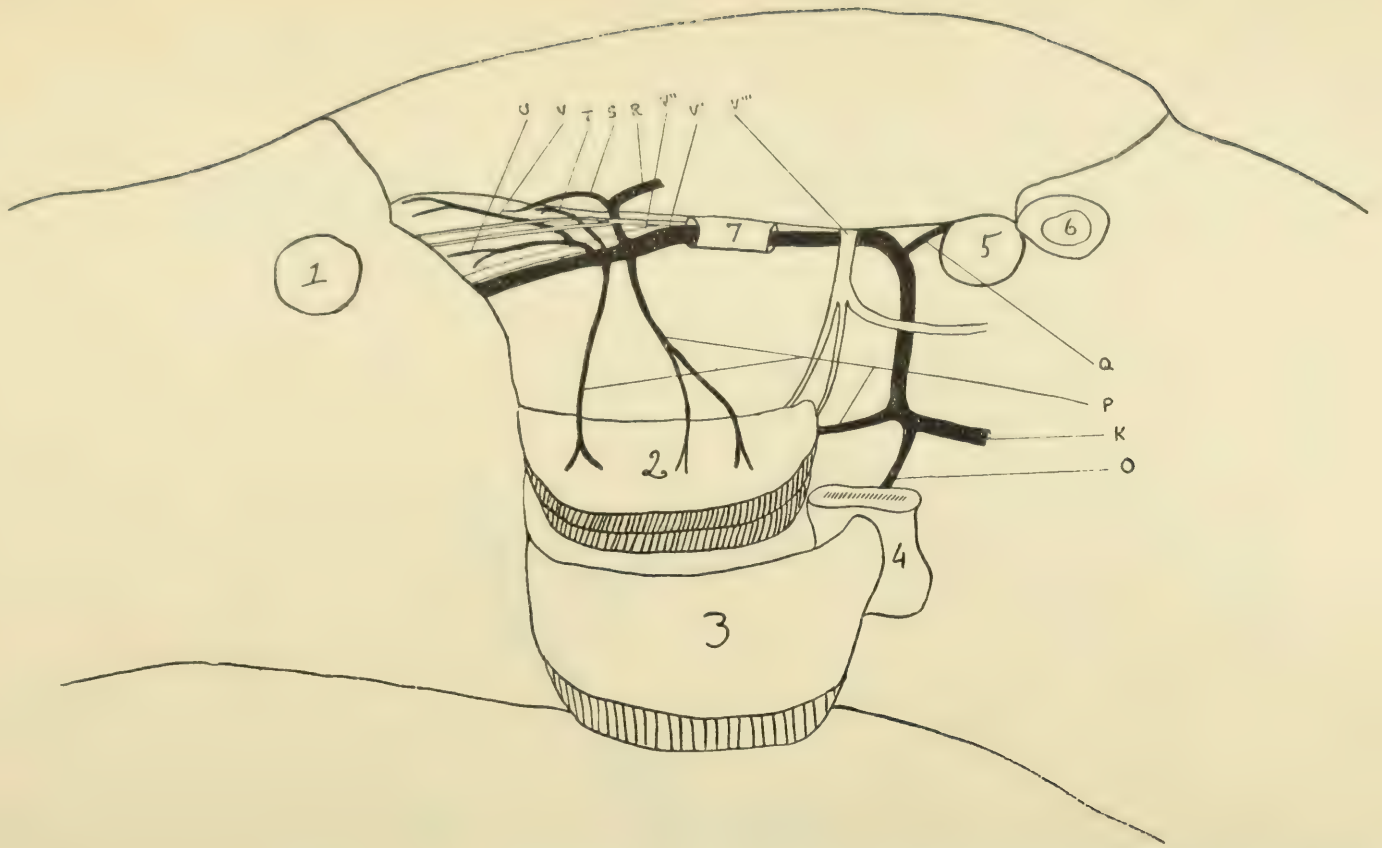
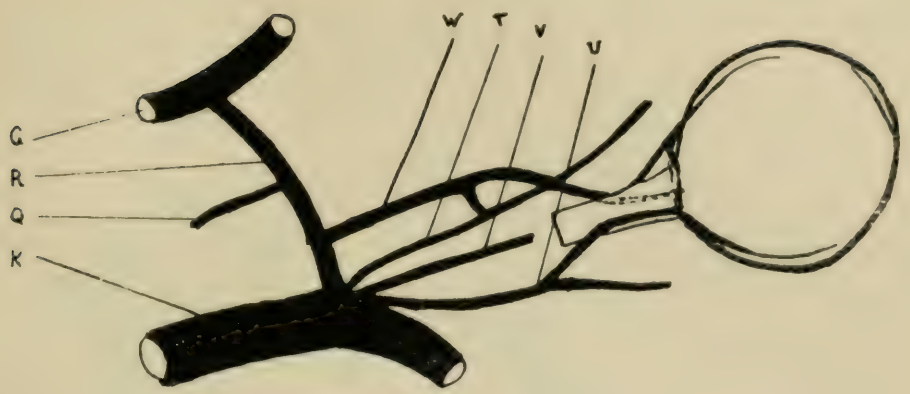
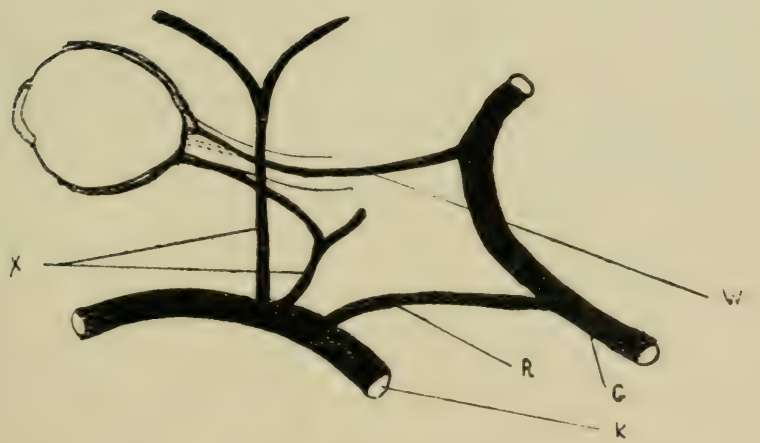


FIG. 5.—Scheme of internal maxillary and ophthalmic arteries in the dog:—K, internal maxillary artery; O, inferior alveolar artery; P, deep temporal artery; Q, middle meningeal artery; R, ramus anastomoticus; S, superior ophthalmic artery (inferior of Bellarminow); T, ethmoidal artery; U, lacrymal artery; V, frontal artery. V′V″V‴, the three parts of the fifth nerve. 1, placed on the eye; 2, on the temporal and pterygoid muscles, cut and turned down; 3, on the masseter, also turned down; 4, on the lower jaw; 5, on the bulla





FIGS. 6, 7, and 8.—These represent variations in the orbital arteries in the dog:—G, internal carotid; K, internal maxillary artery; Q, middle meningeal artery; R, ramus anastomoticus; W, superior ophthalmic artery; X, inferior ophthalmic arter T, ethmoidal artery; U, lacrymal artery: V, frontal artery.

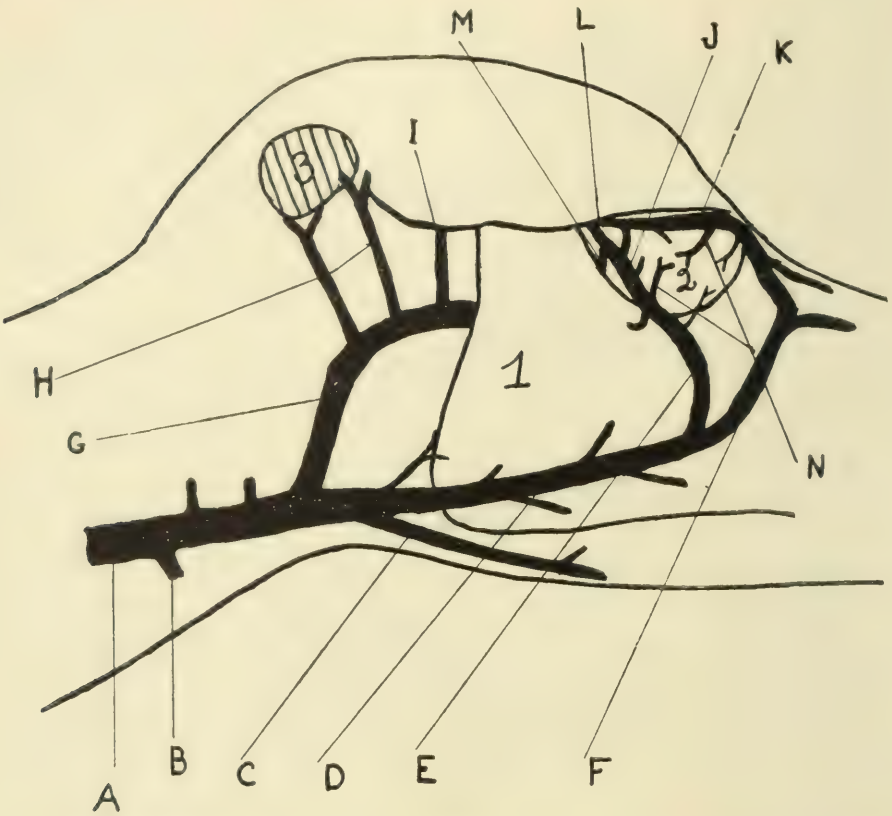


FIG. 9.—Scheme of the external jugular and ophthalmic veins in the dog; the eyeball, margin of the orbit, and masseter have been removed:—A, external jugular vein; B, communication to external jugular of the opposite side; C, lingual; D, common facial; E, deep facial; F, superficial facial; G, internal maxillary; H, auricular; I, to lateral sinus; J, inferior ophthalmic or ophthalmo-cerebral vein; K, superior ophthalmic or cerebro-facial vein; L, to cavernous sinus; M, to infraorbital vein; N, vorticose veins. 1 is placed on the inferior maxilla; 2, on the floor of the orbit; 3, on the ear.

ON THE RELATIONS BETWEEN INTRAOCULAR TENSION AND THE GENERAL BLOOD-PRESSURE.

By J. Herbert Parsons, F.R.C.S., and Thomas Snowball, M.B., Ch.B. (Aberd.).

(From the Physiological Laboratory, University College, London.)

In bringing forward these experiments, we do not propose to discuss the results obtained by earlier observers, as many of these are contradictory, owing to faulty methods and other causes. Our experiments most accurately conform to those of von Schultén* and Bellarminow,† although, as will be seen later, the method they employed is open to criticism.

Method.

About 40 experiments have been performed—upon dogs and a few cats. The animals were anæsthetised with morphia and A.C.E. mixture: in most cases they were also curarised. The blood-pressure was usually taken from the femoral artery, occasionally from the carotid on the opposite side to the eye which was being observed.

The apparatus used for the measurement of the intraocular tension consisted of a cannula (A), a fine-bored glass tube (B), a manometer, and a pressure bottle (Fig. 1). The cannula (A) is a modified form of Leber's intraocular pressure cannula, and is the same as that used by Starling and Pflüger for determining the rate of flow of the aqueous secretion. It consists of a gilt hollow needle, having two small lateral openings at *a* and *b* upon opposite sides near the pointed end, which is also open. This ensures free communication with the interior of the eye. The distal end, *c*, is closed by a plug, which can be replaced by a straight wire

* V. Schultén, Arch. f. Ophth., vol. xxx, part 3, p. 1, 1884.

† Bellarminow, Pflüger's Archiv., vol. xxxix, 1886; Annales d'Oculistique vol. xcvii, p. 181.

for cleaning out any clot or other obstruction. A side tube, *d*, near the distal end, leads off to the manometer by means of two fine-bored glass tubes, *e* and B, connected by india-

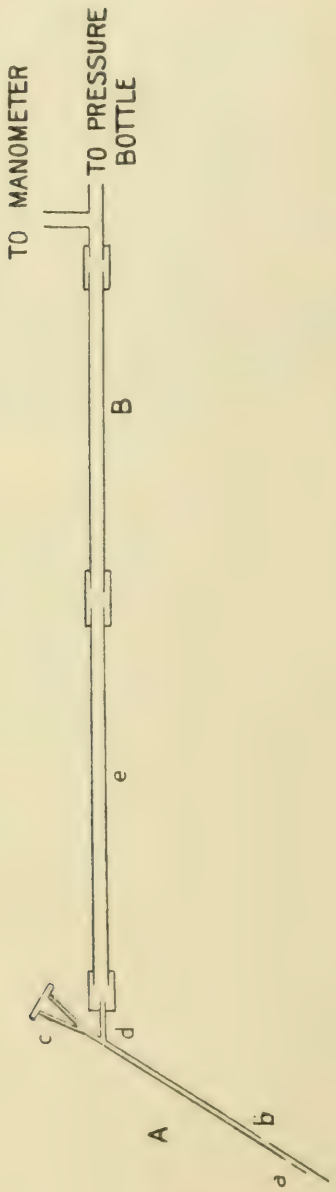


FIG. 1.

rubber joints. The tube *e* is not essential, but is inserted for convenience. The flexible joints allow a limited amount of mobility for the necessary manipulations; they are made as

little elastic as possible by bringing the glass tubes nearly into apposition, and using thick-walled pressure tubing for the junctions. A glass T-tube connects B with the manometer and the pressure bottle.

The manometer used for obtaining graphic records was either a Hürthle manometer, provided with a thin rubber membrane, or an ordinary Marey tambour, about twice the diameter of the Hürthle, with a rather thicker membrane. In most of the experiments only qualitative changes were observed, so that it was unnecessary to calibrate the manometer. In a few cases in which absolute measurements were taken, it was simpler and more accurate to use a fine-bored mercury manometer.

In many of the earlier experiments which were performed by one of us (J. H. P.), the cannula was inserted into the vitreous chamber of the eye. When this method worked well, it gave as conclusive results as in the later experiments, in which the cannula was inserted into the anterior chamber. The former method, however, is not so reliable as the latter, owing to the consistency of the vitreous, which often prevents a free to-and-fro movement in the cannula, and may cause a valvular effect. It is of interest as demonstrating the identity of the results obtained from the two chambers, but those obtained from the anterior chamber are generally the more reliable. Hence it was considered advisable to repeat the experiments of v. Schultén and Bellarminow, in which the cannula was placed in the vitreous.

A further modification of all previous experiments was also introduced. It has been considered essential that no fluid should either leave the eye or enter it when the cannula is inserted. It is, however, quite impossible to accurately foretell the intraocular pressure before inserting the cannula. In our experiments, the whole apparatus was filled with normal saline solution; the pressure bottle was raised to about the pressure anticipated, and the cannula was introduced whilst the fluid was flowing from it. It is very

important that the cannula be perfectly sharp and smooth, so that it may be pushed across the anterior chamber very quickly, otherwise the aqueous escapes, and the iris and lens are wounded and block the apertures in the cannula. Immediately after the cannula is inserted, an air bubble is let into the tube B. The pressure bottle is then raised or lowered according to the movements of the bubble. In a few minutes it assumes a mean position, varying only in accordance to the transmitted cardiac and respiratory waves.

Results.

In determining the effects of changes in the general blood-pressure, it is first necessary to eliminate other possible sources of variation in the intraocular tension. Of these, the most important are the effects of contraction of the extrinsic and intrinsic muscles of the eye. The former consist of two groups—the striated and the unstriated. Contraction of the extrinsic striped muscles, closure of the lids, &c., produce marked changes in the intraocular tension. It is therefore necessary to fully curarise the animal. The extrinsic unstriped muscles have been found to be without effect in the dog, though they are a source of error, as will be mentioned later, in the cat. Movements of the intrinsic muscles of the eye produce no effect upon the intraocular pressure; or, if any, it is too slight to be manifested by the methods employed. Thus the effects obtained in various ways with and without complete paralysis of the iris and ciliary muscle by atropin are identical.

Cervical Sympathetic.—Stimulation of the peripheral end of the cervical sympathetic was investigated in the cat. It was invariably followed by a considerable rise in intraocular pressure, irrespective of any change in the general blood-pressure. This confirms the results obtained by Adamük. v. Hippel, and Grünhagen, &c. It is not due, however, to vascular changes occurring in the eye. It is not due to movements of the iris, since it occurs after prolonged instil-

lation of atropin previous to the operation, and also after large intravenous injections of atropin, sufficient to abolish inhibition of the heart on strong stimulation of the vagi. It was finally proved not to be due to vascular changes, since it occurred for a considerable period after the death of a curarised cat, and is therefore undoubtedly due to pressure exerted upon the globe by the contraction of unstriped muscle contained in the orbit and innervated by the cervical sympathetic. It is therefore impossible to demonstrate by this method any changes in the intraocular vessels by stimulation of the cervical sympathetic. This fact vitiates all Bellarminow's results, which were obtained from cats.

Vth Cranial Nerve.—Stimulation of the peripheral end of the Vth nerve has been without effect, except when a simultaneous rise of blood-pressure occurred, probably from spread of current to meningeal sensory nerves, still connected with the brain. The manometer is probably not sensitive enough to record the variation due to local dilatation, if this occur.

Changes in the Blood Supply of the Eye.—In lower mammals (dog, cat, &c.) the intraocular blood supply is derived almost entirely from the internal maxillary artery, which forms the termination of the external carotid.* In the dog, there is a large anastomotic branch from the internal carotid within the skull, which usually joins the internal maxillary just before the ophthalmic artery or arteries are given off. Tying the external carotid, therefore, cuts off the main blood supply to the eye. In spite of the slight rise in general blood-pressure which this operation causes, there is invariably a well-marked fall in intraocular tension. Ligature of the internal carotid, which is much smaller than the external, causes no change in intraocular tension if the external is still patent. If the external carotid is tied, although the intraocular tension falls, and remains subnormal for a considerable period, the eye is still well supplied with blood, through the internal, by its anastomotic

* *Vide* Henderson, *ante* p. 260.

branch, and through circle of Willis; so that changes in the general blood-pressure still affect the eye.

Ligature of the common carotid of the opposite side causes no effect.

Changes in General Blood-Pressure.—The intraocular tension responds passively to all variations in the general blood-pressure. This is the case also if the external carotid is tied, or if the skull is freely laid open.

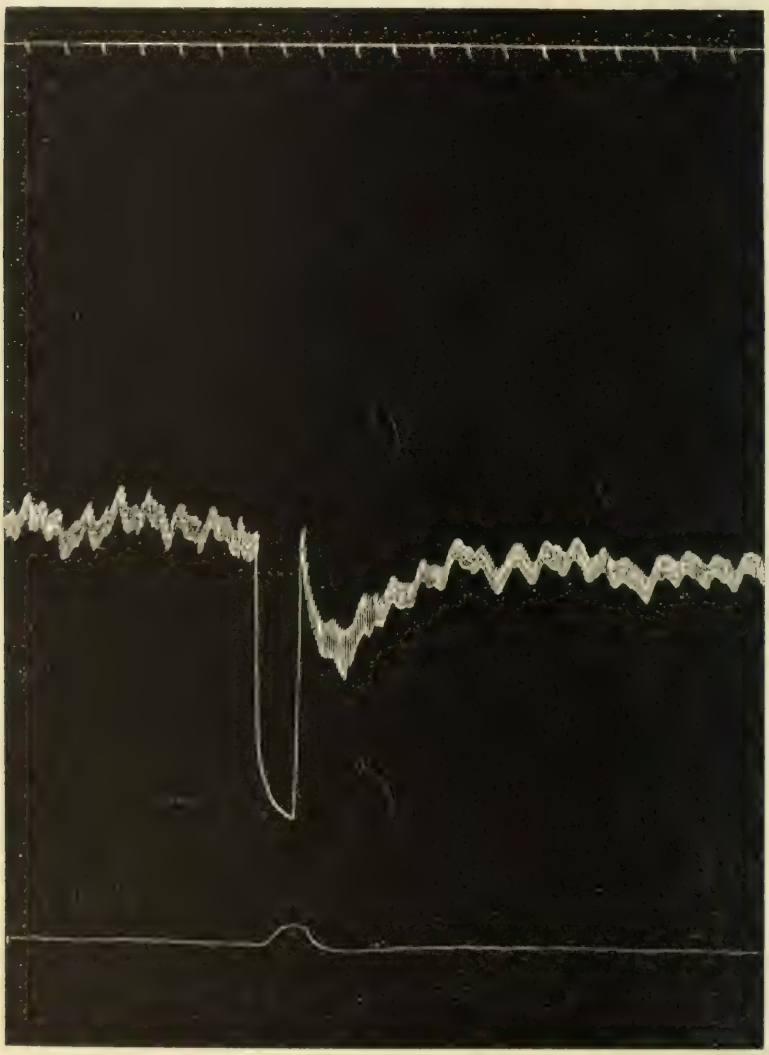


FIG. 2.—Dog. Morphia, A.C.E. and curare. Femoral blood-pressure. Compression of thoracic aorta. Time in 10 sec. intervals.

The respiratory and cardiac oscillations are usually clearly represented upon the air bubble in the tube B: they are not recorded by the manometer. The general curve of

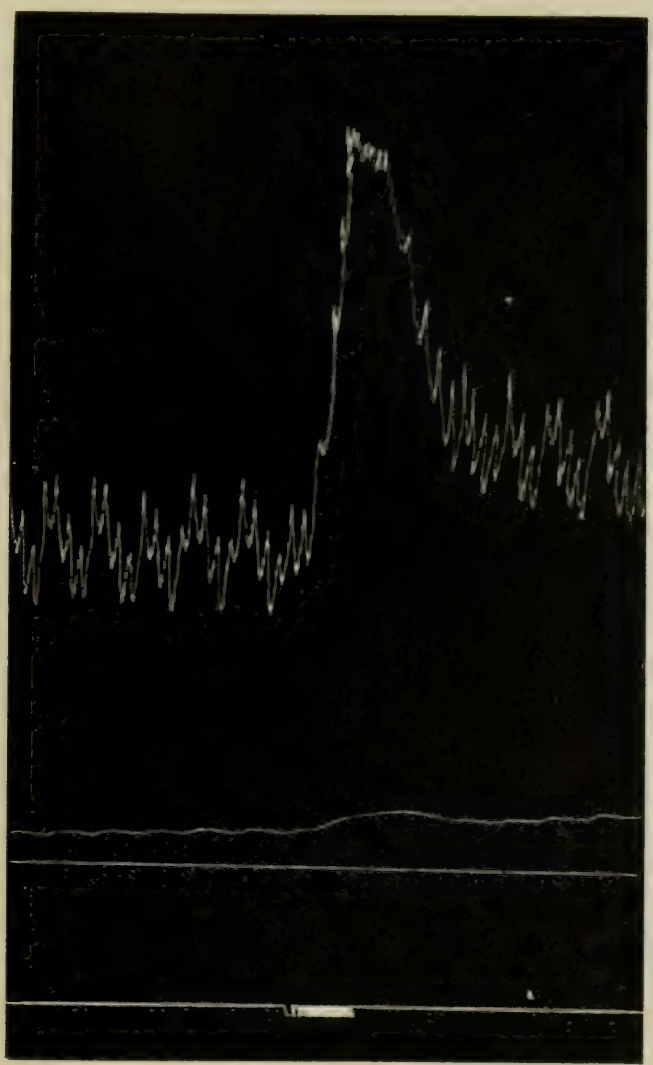


FIG. 3.—Dog. Morphia, A.C.E. and curare. R. carotid pressure; L. intra-ocular pressure. Traube-Hering curves: stimulation of vaso-motor centre.

the blood-pressure is reproduced by the intraocular manometer, but, as it were, damped. There is a short latent

period (1—2 sec.), the changes are slower, and the curve flatter.

The blood-pressure has been varied by the following means.

(*a*) *Mechanical.*—Rise of blood-pressure in the anterior part of the body has been brought about by compression of the abdominal or thoracic aorta. It is accompanied by a passive rise of intraocular tension.

(*b*) *Stimulation of Nerves.*—Stimulation of the central end of sensory and mixed nerves (*e.g.*, sciatic) causes rise in both. The central end of the vagus has always given us a pressor effect, accompanied by rise in eye-tension. Cardiac inhibition through the peripheral end of the vagus causes a sharp fall in intraocular pressure. The rise in blood-pressure by stimulation of the vaso-motor centre, peripheral cut end of cervical cord, or splanchnic nerves, causes the usual passive effect. Traube-Hering curves are accurately reproduced.

(*c*) *Drugs.*—Adrenalin, nicotin, strychnin, pilocarpin, quinin, amyl nitrite and large doses of chloroform give results in accordance with their effects upon the blood-pressure. It may be noted that pilocarpin apparently has no specific secretory effect. We have not been able to obtain any local vaso-constrictor effect by injecting adrenalin into the common carotid artery.

(*d*) *Asphyxia.*—The rise in blood-pressure due to asphyxia is accompanied by a corresponding rise in intraocular tension, which continues during the fall in blood-pressure. This is doubtless due to the obstruction to the return of venous blood to the heart in this stage.

(*e*) *Death.*—The normal intraocular pressure of the dog varies from 20 to 30 mm. Hg. After death it gradually and very slowly falls to zero.

We have done some experiments in which the intracranial pressure has been raised by various means. If air is pumped into the cranium, the animal soon dies from absorp-

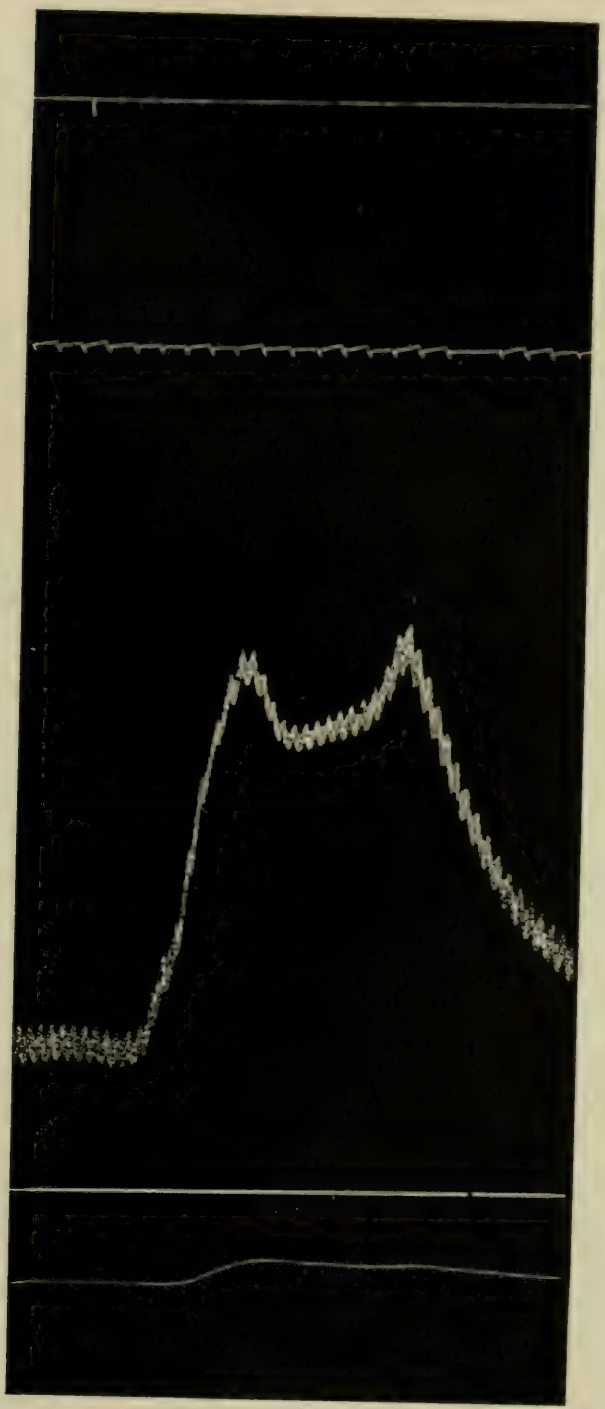


FIG. 4.—Dog. Morphia, A.C.E. and curare. Femoral blood-pressure; external carotid tied. 1 minim of 5 per cent. solution of nicotin injected into femoral vein. Time in 10 sec. intervals.

tion of air into the circulation. Under these circumstances the intraocular pressure rises enormously, owing to the same cause as in the later stages of asphyxia, but the effect is much more marked.

Remarks.

In interpreting the results obtained by the manometric method upon the eye, we have to consider—apart from the accuracy of the method itself—(1) the elasticity of the coats of the eyeball, (2) vaso-motor changes, (3) the secretion of intraocular fluid.

Koster Gzn,* experimenting upon excised eyes, showed that, under low pressures, up to 100 mm. Hg, the energy is expended in altering the shape of the eye, so that it tends to assume a spherical form; whilst it is only under pressures above 100 mm. Hg, that the elasticity of the sclerotic becomes the essential factor. Hence for the comparatively small variations at low pressures now under consideration, we may ignore the elasticity of the sclerotic.

As regards the secretion of intraocular fluid, Starling and Pflüger have shown in some experiments which will be published shortly, that the secretion follows the physical laws of filtration. In any case, it is improbable that active secretion is so rapid as to affect much the intraocular tension during the comparatively rapid changes brought about in these experiments.

Consequently, the variations observed may be regarded as due to changes in the volume of the intraocular blood-vessels: even if slight changes in secretory activity occur they will be in the same direction, constriction of arterioles being associated with diminished, and dilatation with increased secretion. The evidence of active vaso-motor changes in the eye have hitherto depended entirely upon ophthalmoscopic

* Koster Gzn, Arch. f. Ophth., vol. xli, part 2, p. 141, 1895; vol. lii, part 3, p. 402, 1901.

observations, which are open to grave doubt. We have ourselves failed to demonstrate such changes by the present method; but the experiments afford ample evidence of passive vascular changes.

The expenses of this research were defrayed by a Royal Society grant.

METASTATIC CARCINOMA OF THE CHOROID.

By J. Herbert Parsons, *Curator*.

The following are the detailed clinical and pathological notes of the case of metastatic carcinoma of the choroid referred to by Mr. Holmes Spicer on p. 235 of the Transactions of the Ophthalmological Society of the United Kingdom, vol. xxii, 1902:—

The patient was a lady, æt. 37, unmarried. Her right breast was removed in 1899 for scirrhus, a large area of skin and some axillary glands having been taken away at the operation. The patient enjoyed good health afterwards until May 14th, 1902, when she consulted Mr. John Couper on account of dimness of vision in the L. eye, noticed a few days previously. V. in this eye, after correction of slight hypermetropic astigmatism = bare 6/9, J 1 slowly, R. V. corrected = 6/4, J 1.

L. Ophthalmoscopic Examination.—Media clear = growth clearly seen as an opaque detachment up and in from the disc. Some of the adjoining retina below was detached in broadish folds, and had remained more or less transparent, as did the fluid between it and the choroid. There was nowhere any trace of hæmorrhage visible.

Later on the same day a consultation was held with Mr. Howard Marsh, and on the following day with Mr. Holmes Spicer.

The eye was enucleated on May 16th, 1902.

[I am indebted for the clinical notes and permission to publish the case to Messrs. John Couper and Holmes Spicer.]

Pathological Examination.—The eye was placed in 10 per cent. formol immediately after excision. After a few days it was frozen, and bisected sagittally with a slight inclination down and out.

Macroscopic Examination.—Cornea, a.c., iris and lens normal. Retina detached widely below, with albuminous coagulum in

FIG. 1. × 4.

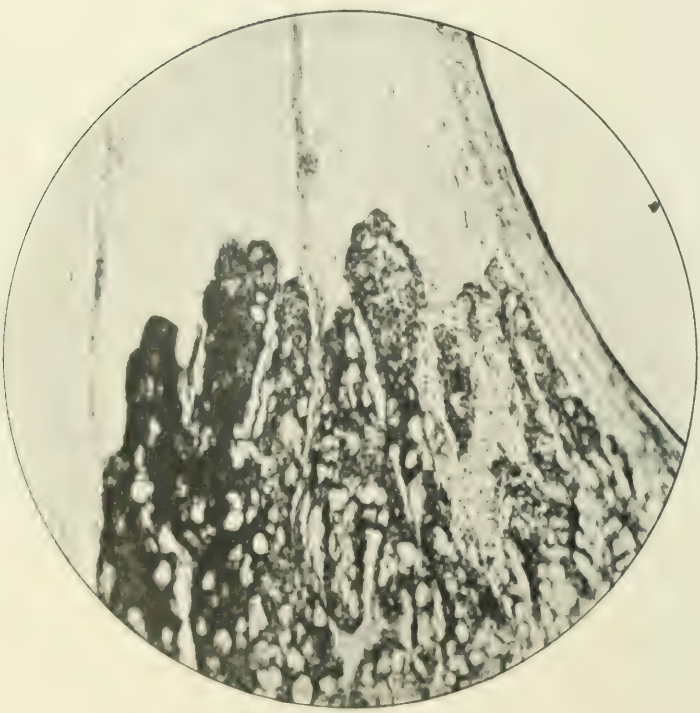


FIG. 2. × 60.

PARSONS—Metastatic Carcinoma.

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the sub-retinal space. Above it is still in apposition to the growth. Choroid shows large thin whitish growth, extending forwards from the disc to the equator. It is barely 2 mm. thick in its thickest part, which is near the front. It extends over a large area of the choroid.

Microscopic Examination.—Cornea, a.c., iris and lens normal. The retina is detached widely below, but is adherent to the growth, except over its peripheral half, where it is detached. (The latter may be an artefact.) The growth extends widely over the lower side, starting from the edge of the disc. There is also a patch, 4 mm. broad by 1 mm. thick in the thickest part, situated above the disc. It also invades the lower part of the nerve, extending for about 1 mm. behind the lamina cribrosa. (Fig. 1.)

The growth consists of spheroidal epithelial cells, many of which have dropped out, whilst many others appear vacuolated. They have an alveolar arrangement, and there is no stroma between the individual cells. The alveoli form spaces bounded by the separated fibres of the choroid. At the anterior edge the phalanx of columns of the growth advancing between the planes of the choroidal stroma is particularly striking (Fig. 2). Minute outlying nodules are seen, few in number, in the sclerotic.

The growth is a glandular carcinoma. It contains less fibrous tissue than the primary scirrhus contained (presuming it to have been a typical example). This is not unfrequent in metastases.

Remarks.

This is a typical metastatic carcinoma of the choroid derived from a primary growth in the most common situation.

In the discussion upon Mr. Rockliffe's case (Trans. Ophth. Soc., vol. xxii, p. 236, 1902), Dr. Hill Griffith called attention to the flatness of these tumours as compared with primary sarcomata of the choroid. Mr. Devereux Marshall remarked that "sarcomata, if seen early enough, were doubtless flat." I do not think that this statement is

generally true, if by "flatness" we understand a large difference between length and thickness. This is universal in metastatic carcinomata of the choroid at all stages of their growth. It is only rarely the case in sarcomata, such growths having been described in German ophthalmic literature as "Flächensarcome." They are extremely rare—only one example having been recorded in the pathological reports of this hospital. They are allied to the Annular Sarcomata ("Ringsarcome") of the ciliary body, an infiltrating type of growth, of which I am describing a good example in a forthcoming number of Graefe's Archiv für Ophthalmologie. The ordinary primary sarcoma of the choroid, even from the earliest stages observed in typical examples, is lenticular in shape, and therefore cannot be accurately described as flat.

The shape of these tumours is more important than might appear at first sight, since it indicates fundamental differences of origin and growth. The ordinary primary sarcoma starts in the actual stroma cells of the choroid itself. Following the laws of growth, the cells proliferate in the directions of least resistance. Since the growth commences *in* the actual tissues themselves, the resistance is equal in all directions at first, so that at this stage we might expect the tumour to be spherical; such an early stage, however, has never been described. Soon the greater resistance on the side of the sclerotic makes itself felt, the advance is less in this direction, whilst equal in other directions; hence the tumour becomes roughly elliptical, growing twice as fast laterally as in thickness. This mode of advance continues until the membrana vitrea is burst through, which results from equable stretching rather than from any localised attack by the growth upon any one spot. At the hole in Bruch's membrane, which is necessarily central as to the surface of the tumour, resistance is greatly relieved; proliferation becomes most active in this direction, and a new focus is established in the sub-retinal space. Here again resistances are equalised, growth proceeds equally in

all directions, and the very characteristic globular head of the tumour is formed, connected with the main mass by a short neck at the level of Bruch's membrane.

The commencement and sequence are quite different in the case of metastatic carcinomata of the choroid. Metastasis here can only occur through the blood-vessels, since it is inconceivable that deposits can be brought from the breast, for example, by way of lymphatics. The frequent affection of both eyes is almost conclusive as to a blood-vascular origin. The earliest focus must be of the nature of an embolus, probably in the finer arterioles of the choroid, The vessel affected is blocked, the cancer cells proliferate, and burst through, or replace the walls of the vessel. They are now situated in the lymph-spaces in the choroid, which lie directly outside the blood-vessels. Hence they lie *between* the planes of the choroidal stroma. We know from the frequent cases of detachment of the posterior part of the ciliary body, that traction readily draws the planes of the stroma of the uveal tract apart, and that those which offer greatest resistance and persist longest are those which are more or less concentric with the curvuture of the globe, those transverse or oblique to this direction being relatively feeble. The same is shown in the further growth of metastatic deposits. Lying as the cells now do between these concentric planes, the directions of least resistance are along them; hence most of the growth is lateral, a minimum increase of thickness taking place. The appearances shown in Fig. 2 are, to my mind, most striking, and afford overwhelming evidence of the accuracy of this view as to the mode of growth. Whether the same explanation applies to the rare "Flächensarcome" or not, the scantiness of the material makes it difficult to decide.

Of course, it is to be understood that these descriptions apply to the essential tendencies of growth in the given cases. These tendencies are often modified in detail, *e.g.*, by proximity to a vorticose vein, so that a new path of diminished resistance is open to the growth. Such modifi-

cations are usually obvious in individual cases, and do not affect the general statements made above.

Since the publication of Mr. Devereux Marshall's excellent paper on "Metastatic Carcinoma of the Eyeball" in an earlier number of these Reports (vol. xiv, pt. 3, p. 415,

No. of case.	Author.	Where published.	Sex.	Age.	R. or L.	V. and T.
25	Samelsohn ..	Berlin klin. Woch., 1891	F.	43	L.	—
26	De Schweinitz ..	Trans. Amer. Ophth. Soc., 1898, p. 313	F.	40	R.	V = fingers at 2 ft. Tn.
27	De Schweinitz ..	Ditto	F.	43	L.	V = 10/200
28	Von Michel ..	International Congress, Utrecht, 1899	F.	—	—	Tn... ..
29	Rowan	Trans. Ophth. Soc. U.K., 1899	M.	55	L.	—
30	Lagrange ..	Tumeurs de l'œil, vol. i, p. 512	F.	48	R.	T + 1 ..
31	Lagrange ..	Ditto	M.	—	R.	—
32	Rockliffe ..	Trans. Ophth. Soc., 1902	F.	46	L.	V = p.l. .. Tn.
					R.	V = 6/6 .. Tn.
33	Parsons	Present paper	F.	37	L.	V = 6/9. .. Tn.

1897), 8 fresh cases have now been published; and another earlier case, which Mr. Marshall missed, brings the total number of cases up to 33, 41 eyes being affected.

The following table supplements Mr. Marshall's, and brings it up to date :—

Pathological examination of eyeball.	Seat of primary growth.	Seat of secondary deposits other than eye, and result of post-mortem, if obtained.	Duration of life after eye was first affected.	Remarks.
Flat tumour extending over disc, invading nerve. Extraocular tumour also	—	No autopsy.		
Flat tumour, extending over macular region nearly to equator	L. breast ..	? Intracranial and spinal.		
Flat tumour, extending over macular region, with red spots	L. breast ..	—	—	Clinical only.
Flat tumour over temporal side	Breast.			
Flat tumour, covering posterior pole	Lung.			
Flat tumour over whole temporal and half nasal areas, with growth at outer part of iris and in a.c.				
Intra- and Extra-ocular tumours. Intra-, pigmented anteriorly; extra-, quite white. 'Undoubted carcinoma.'				
Flat tumour over ¾ fundus, extending to ciliary body one side; O.N. involved.	R. breast ..	Pleurisy. Tumour in R. groin.	9 months.	
Not examined	—	No autopsy ..	—	Clinical only.
Flat tumour from disc to equator, up and in; small down and out; O.N. involved	R. breast.			

TWO CASES OF GUMMA OF THE CILIARY BODY.

By J. Herbert Parsons, *Curator*.

Cases of gumma of the ciliary body rarely become available for pathological examination. I have had the unusual opportunity of examining three very severe, apparently uncomplicated cases, recently. One of these is reported by Mr. Morton and myself in the last volume of the Transactions of the Ophthalmological Society (vol. xxii, p. 266, Plate xxii, Fig. 2). It was removed from the cadaver. The other two are recorded here; the disorganisation of the eyes was so great, and the pain so severe that it was considered advisable to excise them.

Case I.

T. H., male, æt. 64.

3.vii.02. History of primary sore 3 months ago. Scaly papular rash on forehead, rupial on body.

L. eye. Chemosis: cornea hazy; iritis, posterior synechiæ.

17.vii.02. Lymph in a. c.; blood in substance of cornea; sclerotic bulged in two places below cornea by ? gumma.

24.vii.02. L. V. – bare p. l., projection bad.

30.vii.02. Yellowish projection from sclerotic in ciliary region below; exudation in lower part of a. c.

Pathological Examination.—The eye was hardened in 10 per cent. formol, frozen, and bisected in a sagittal plane.

Macroscopic Examination.—Cornea—surface rough, looks blood-stained. A. c.—full of exudate and blood. Sclera—perforated below by inflammatory mass, continuous with the ciliary body, and communicating with a. c.; the mass is 5 mm. thick in thickest part (Fig. 1). Lens—cataractous. Vitreous—filled with white filaments which radiate from the optic disc and also from the gumma. Retinal and choroid *in situ*.

Microscopic Examination.—Cornea—epithelium irregular, somewhat œdematous; Bowman's membrane intact; substantia

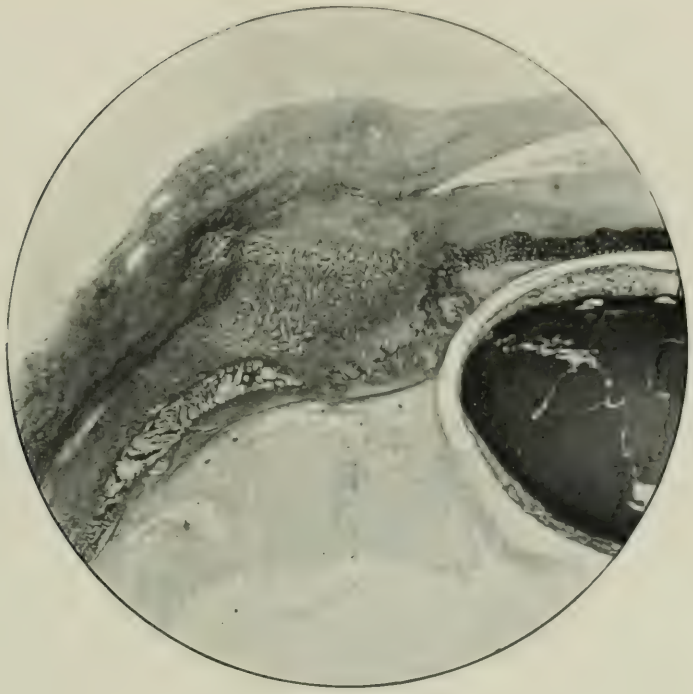

FIG. 1. × 6.

FIG. 2. × 7.

PARSONS—Gumma of Ciliary Body.

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propria slightly infiltrated, especially at periphery; Descemet's membrane intact, endothelium mostly disappeared, the membrane being covered with red corpuscles and exudate. A. c.—full of fibrinous coagulum containing leucocytes and red corpuscles; the upper angle is blocked with fibrin and cells, canal of Schlemm being open; lower angle is full of fibrin, &c., continuous with iris tissue and ciliary body. Iris—much inflamed, swollen and infiltrated throughout, especially below, where it is filled with fibrin and inflammatory products, which are continuous with those in the a. c.; retinal pigment epithelium layer enormously thickened, especially below. Posterior chamber—full of fibrinous exudate. Ciliary body—inflamed and infiltrated with round cells above; below it is the seat of a necrosing granuloma, almost identical in every respect with that described by me in Trans. Ophth. Soc. U. K., vol. xxii, p. 266 (with Plate xxii, Fig. 2). There is the same massing of inflammatory products of the same type, without giant-cells, accompanied by extensive necrosis and destruction of the sclerotic. Vitreous—pervaded by fibrinous inflammatory products, which especially surround the ciliary processes, the latter being almost depigmented. Lens—it is doubtful if the capsule is intact; cortical fibres are vacuolated; nucleus normal. Retina—much infiltrated and disorganised, the layers being differentiated with difficulty. Choroid—congested and inflamed.

Case II.

C. G., female, æt. 25. L. eye quite normal 6 weeks ago; then became watery and slightly bloodshot. There was pain in the side of the face. Three weeks ago the inflammation increased, and the vision rapidly failed. The "staphyloma" was first noticed a week ago. The patient has been losing flesh for 6 weeks; no history of syphilis.

R. eye. Cornea clear. A. c. good. Iris and pupil normal. Fundus normal. V = 6/5. Tn.

L. eye. Intense injection. "Staphyloma" below cornea, containing ciliary body; sclerotic thinned here. Cornea hazy. A. c. shallow. Iris discoloured; pupil blocked, margin bound down to lens. No red reflex. V = hand shadows, projection bad. T slightly less than R.

On the following day the L. T = −2, and it was noticed that the corresponding pre-auricular gland was swollen.

The eye was excised 5 days after first note.

Pathological Examination.—The eye was hardened in 10 per cent. formol, frozen, and bisected in a sagittal plane.

Macroscopic Examination.—Cornea hazy. A. c. of unequal depth; deeper below where the iris is retracted, shallow above, where the angle is blocked by adhesion of the iris to the back of the cornea for 3 mm. Iris—pupil very small, blocked; ring synechia; iris bombé above; in a condition of total posterior synechia below. Lens, *in situ*. Ciliary body—much thickened below; covered internally by dense white exudate, which stretches out into the vitreous; there is a brownish swelling outside in the situation of the "staphylomatous" condition: greatest thickness = 4·5 mm. (Fig. 2). Vitreous—full of exudates. Retina and choroid—*in situ*.

Microscopic Examination.—Cornea—epithelium and Bowman's membrane intact and normal; substantia propria slightly infiltrated throughout; Descemet's membrane normal above. A. c. —upper angle blocked by adhesion of iris to cornea for 3 mm.; lower angle widely open where the gumma invades a. c. Iris—intensely congested and inflamed throughout; bombé above—there are small dense aggregations of mononuclear leucocytes at the periphery in this part, and both the retinal and uveal pigment are much disturbed and have proliferated; adherent to lens over whole area below, with the intervention in places of leucocytes and low developed fibrous tissue. Ciliary body—congested above, and slightly detached in posterior part, where there is an albuminous coagulum in the subciliary space. Below it is the seat of a necrosing granuloma, which has broken through into the a. c. in front, and into the vitreous internally, so that it is in contact with the lens just in front of the equator. The ciliary processes are entirely disorganised, and are only represented by irregular wavy lines of persistent retinal pigment cells. The main mass is made up of badly staining leucocytes, which have run together into an albuminous mass, in which faint remnants of nuclei, red blood corpuscles, shreds of fibrous tissue and pigment granules can be seen. There are no giant-cells, and there is no effort towards repair. The sclerotic

has been destroyed for 3 mm., the lamellæ at its termination being spread out, and separated from one another by leucocytes. The external mass, which looked brown macroscopically, consists of intensely inflamed episcleral tissue. The blood-vessels are widely dilated and crammed with red corpuscles; the stroma is densely packed with mononuclear leucocytes. Lens—capsule intact; the central part is homogeneous. Vitreous—full of fibrinous exudates, especially below; the interstices of the network are filled with leucocytes of various kinds, but mainly polymorphonuclear. Retina—inflamed everywhere; much infiltrated with round cells, especially near the gumma; the vessels are all widely dilated, full of red corpuscles, and surrounded by dense leucocytic infiltration; rods and cones disorganised everywhere, except near posterior pole; nuclear layers retain a quasi-normal appearance. Choroid—much congested and inflamed: there is a small hæmorrhage below, just behind the gumma. Optic disc—swollen and infiltrated. Optic nerve—looks normal.

Remarks.

The subject of gumma of the ciliary body was briefly discussed in the paper in the Transactions of the Ophthalmological Society, with a review and bibliography of the literature. There is little to add here other than to say that these cases support the conclusions arrived at there, especially as regards the microscopic identity of the so-called "papillomata" (*i.e.*, condylomata) with gummata of the ciliary body, and also as regards the absence of grant-cells in these syphilitic granulomata in this region. The diagnosis of syphilis is undoubted in Case I, though more stress is to be laid upon the rash than the history. Continental authors (*e.g.*, Ostwalt) would almost certainly class this as a condyloma, in spite of the fact that it is essentially identical microscopically with the gumma recorded in the Transactions. Whether they would class Case II with the condylomata or gummata is uncertain, and, as we have seen, quite immaterial.

A NOTE ON THE USE OF HAAB'S MAGNET.

By William Lang.

When a foreign body has been drawn forward from the vitreous chamber in the usual manner, by Haab's magnet, it may become entangled in the iris, and remain in the posterior chamber, instead of passing through the pupil to the back of the cornea, where it can drop into the bottom of the anterior chamber when the current is cut off.

When the fragment is in the anterior chamber, it can be removed through a corneal section without disturbing the iris, but as long as it is in the posterior chamber, its direct extraction has necessitated the tearing through or cutting away of a piece of the iris. To avoid this, I have used the following plan:—As soon as the foreign body, attracted by the magnet, is seen to be lifting the iris forwards, the current is cut off and the patient is placed on the operating table, which is raised to a height convenient to the surgeon. The speculum is introduced and a transverse puncture 3 or 4 mm. wide is made midway between the centre and the periphery of the cornea at a point opposite to the situation of the foreign body. If the broad needle is held with its blade in the same plane as the iris, the section can be made and the needle withdrawn without loss of aqueous. A steel spatula, made for me by Weiss, in the form of a spud, but with a rounded end, is passed between the lips of the corneal section across the anterior chamber, through the pupil and between the iris and the anterior lens capsule.

An assistant now connects the base of the spatula, by means of a few strands of soft iron wire, twisted into a rope, with Haab's magnet, which is brought close to the patient's eye; the spatula is then withdrawn with the foreign body adhering to its end. In this way Haab's magnet is made to do, in a more convenient manner, the work of the small

magnet; the iris is not bruised, torn or cut, and with reasonable care, the lense capsule is not damaged.

The flexible rope of very fine iron wire, consisting of about twenty strands 12 inches in length, is connected by means of a clamp with the magnet. If the iron wire is kept in a bottle of absolute alcohol, it remains bright and free from rust.

The Royal London

Ophthalmic Hospital Reports.

Vol. XV. Part IV. October, 1903.

I. SOME REMARKS ON THE USE OF MALLORY'S PHOSPHOTUNGSTIC ACID HÆMATOXYLIN, AND A NOTE ON THE MUSCULUS PAPILLÆ OPTICI OF NIKOLAÏ.

By Frederick Herman Verhoeff, A.M., M.D., Pathologist, Massachusetts Charitable Eye and Ear Infirmary; Assistant Ophthalmic Surgeon, Carney Hospital, Boston, U.S.A.

Phosphotungstic acid hæmatoxylin was brought forward by Mallory* as a differential stain for the neuroglia of the central nervous system, for which purpose it is probably unexcelled. During the past three years the writer has made extensive use of it in staining sections of the eye, and has found it of great value, not alone for neuroglia, but for other tissues as well. It was found, however, that the method of fixation, recommended by Mallory for the central nervous system, was of little service for the neuroglia of the eye, and that in the case of other tissues the results obtained varied with the method of fixation employed. The stain itself, as given by Mallory, is as follows:—

Hæmatoxylin	0·1
Water	80
10 per cent. aqueous solution of phosphotungstic acid (Merck)	20
Peroxide of hydrogen (U.S.P.)	2

* Journal of Experimental Medicine, 1897, p. 532; *ibid.*, 1900, p. 15.

"Dissolve the hæmatoxylin in a little water by the aid of heat and add it, after cooling, to the rest of the water and the acid. The solution keeps perfectly and can be used repeatedly."

This solution, even when used alone, gives excellent results, but, according to Mallory, these are not permanent, and he, therefore, advises the use of an alcoholic solution of ferric chloride to fix the stain. The latter solution removes the pink colour from the connective tissue, or, if the staining solution is old, converts it into a brown colour. The connective tissue, however, may be readily stained again by acid fuchsin alone or by van Gieson's stain. In fact, the writer has found this the best method of using van Gieson's stain for tissues fixed in Zenker's fluid or alcohol. The method of staining sections is given below. Lugol's solution, followed by alcohol, is, of course, to be used only for specimens fixed in Zenker's fluid. If the sections are overstained, the excess of stain can be removed by prolonging the treatment with the ferric chloride solution:

(1) Lugol's solution, 5 minutes.

(2) 95 per cent. alcohol until yellow colour is removed.

(3) Water (to remove alcohol).

(4) Phosphotungstic acid hæmatoxylin, 24 hours or longer.

(5) Wash quickly in water.

(6) 30 per cent. alcoholic solution of ferric chloride, 5 minutes.

(7) Wash in two or three changes of water.

(8) 2 per cent. acid fuchsin or van Gieson's stain, 5 minutes or longer.

(9) 95 per cent. alcohol, two changes.

(10) Ol. origanum cretici.

(11) Xylol balsam.

As just stated, the method of fixation recommended by Mallory does not give successful results in the case of the eye. The fixation methods found most useful by the writer are as follows:—

(1) *Formaldehyde 4 Per Cent., Four Days or Longer.*—Sometimes this gives beautiful results with the neuroglia of the optic nerve and retina, but more often that of the latter fails to strain. Why Müller's fibres should stain intensely in some specimens and remain colourless in others that have been subjected to the same treatment, is difficult to understand. A striking as well as a constant staining reaction after this method, however, is exhibited by the rods and cones, the inner limbs of which always take a strong blue colour, while the outer limbs usually do not stain. Not infrequently the nuclei and fibrils of the rods and cones also stain intensely. This produces a remarkable differentiation between the outer and inner nuclear layers, the latter remaining unstained. The contrast between the two layers may be further heightened by staining 24 hours in lithium carmine after differentiating in the ferric chloride solution. The elements of the internal nuclear layer then take the carmine stain. In some specimens only portions of the retina show this differentiation. It seems to occur most often in cases of separation of the retina or choroiditis, where, of course, the nourishment furnished the retina by the choriocapillaris is interfered with, but it also occurs in apparently normal retinæ. The condition is present also in the retina of the guinea pig and in that of the frog. In the one specimen of the retina of a rabbit, upon which the stain was tried, neither of the two nuclear layers took the stain, but the rods and cones were stained as in the human retina.

These staining reactions seem to show very conclusively that the rods and cones, as well as their nuclei and fibrils, differ essentially from the bipolar cells of the retina. It is not safe, however, to conclude that they are similar in nature to neuroglia. Under any circumstances phosphotungstic acid hæmotoxylin is not such a strictly differential stain as to prove that any substance coloured by it is neuroglia. It is differential, however, in the sense that it distinguishes neuroglia fibrils (not cells) from all other fibrils for which

they are likely to be mistaken. More than this cannot be expected of any so-called differential stain.

Formaldehyde fixation also gives other interesting results with this stain. It is the best method for demonstrating fibrin, for although fibrin itself stains after the other methods of fixation, by this method it is brought out in sharper contrast with the surrounding tissue. The nuclei of normal tissues generally stain feebly or not at all. This is the case also with the nuclei and cells of glioma retinæ. Sarcoma cells as a rule stain deeply, the nucleoli and nuclear membranes being especially well brought out. The nucleoli stain perfectly black, and in some cases are the only parts of the cell to take the stain. The nuclei of cells in an early stage of degeneration stain intensely black. After this method, peculiar markings are brought out in medullated peripheral nerves. These were noted by Mallory after his special method of fixation, but are much more sharply defined after fixation in formaldehyde alone. They appear (Fig. 2) as a series of arrow heads, attached to a central core, all pointing in the same direction. They always stain intensely black, even in sections in which neuroglia fails to stain. The writer has found them present in plexiform neuroma of the eye-lids, and they are therefore of some importance from a diagnostic standpoint. The markings apparently correspond in position to the lines of junction of the Schmidt-Lantermanns segments, and it seems not unlikely that they are due to collections of neurokeratin.

(2) *Formaldehyde* 4 *Per Cent.*, 4–10 *Days;* 10 *Per Cent. Nitric Acid*, 48 *Hours; Running Water*, 24 *Hours.*—This is the best method for the neuroglia of the eye, and is practically always successful. The writer has found it also equally as satisfactory for the brain and spinal cord as Mallory's more elaborate method. Müller's fibres are more apt to stain poorly than the neuroglia fibrils of the optic nerve. This is probably due to the fact that the former really consist of the bodies of neuroglia cells. As a rule, better results seem to be obtained with pathological than with

normal retinæ. Separated retinæ yield particularly good specimens, and beautiful specimens can also be obtained showing the proliferation of neuroglia in phthisis bulbi and atrophy of the retina (Fig. 3). The writer has applied it to a number of cases of glioma retinæ with and without rosette formations, but has never been able to find neuroglia fibres which could be regarded as belonging to the tumours themselves.* Neuroglia fibres, however, are sometimes to be found extending out from a portion of retina that has been incorporated in the tumour. Since fibrin and certain products of necrosis stain blue by this method, care must be used not to mistake them for neuroglia. The nuclei of normal cells also stain, so that phosphotungstic acid hæmatoxylin may be used to advantage as a nuclear stain for any part of tissue that has been decalcified by nitric acid.

(3) *Zenker's Fluid*, 24 *Hours*; *Running Water*, 24 *Hours*.—Before placing the globe in this fluid it should be sectioned without freezing in a vertical plane anterior to the ora serrata. Since the retina is attached at the latter situation, air cannot make its way behind it, and it does not become separated. Of course it may later become separated during the steps preliminary to embedding. This is the best method of fixation for general purposes, although alcohol gives results almost as satisfactory. The cells of normal tissues, tumour cells, and the various cells of inflammation, are all perfectly stained. Elastic tissue stains brown, smooth muscle stains deep blue, and is thus sharply differentiated from connective tissue, which is stained bright red by the acid fuchsin. Mallory states that "smooth muscle fibres sometimes stain after Zenker's fluid, but the results are not constant." This probably applies only to tissues obtained at autopsy, which, of course, cannot always be free from post-mortem changes. In the case of enucleated eyes, however, this difficulty is not met with. Possibly also the staining would not be constant throughout a large piece of tissue,

* Specimens fixed by Mallory's method also fail to show neuroglia fibres in these tumours.

but this again is not a difficulty in the case of the eye. At any rate the writer has never known the method to fail to stain the smooth muscle of the eye, including that of its blood vessels. The dilator fibres of the iris, the nature of which has been much discussed, are deeply stained and their structure so well preserved as to leave no doubt that they are smooth muscle fibres. The fibres of peripheral nerves are well stained, but the peculiar arrow-head markings, mentioned above, are absent.

The retina is stained in a beautiful manner after Zenker's fluid, the rods and cones being brought out especially well. This is due, however, as much to the method of fixation as it is to the stain. The inner limbs of the rods and cones are stained blue, the cone ellipsoid staining more deeply than the other portions of the cones. The outer limbs of the rods and cones appear as transparent hollow tubes, whose walls take a blue tint. In case of the cones the membrane forming the walls of the outer limbs is continued over the inner limbs also. Attached to the inner surfaces of the walls of the outer limbs are to be seen numerous granular particles stained slightly red. When acid fuchsin is not used, they stain blue. Sometimes the granules completely fill the outer limbs, or occasionally four or five globular masses make up their contents. At the junction of the inner and outer limbs of a cone there is always a clear unstained zone, which in the short thick cones usually takes the form of a bulbous expansion. Here and there between the rods and cones there is a transparent substance stained red. In places large globules of this material are seen, suggesting that it is the remains of an interstitial substance which has been for the most part dissolved out.

When the nucleus of a cone is situated outside the limiting membrane, as it occasionally is, there is usually a clear crescent-shaped space just ahead of it, suggesting that the nucleus is situated in a vacuole which it does not quite fill. In the specimens of the writer, there seem to be two distinct classes of nuclei situated outside the limiting mem-

brane (vorgelagerte Körner, Greeff). In one class the nuclei always have the clear crescent spaces ahead of them, and are not limited to any special part of the retina, while in the other the crescents are absent and the cones are short and thick. The interesting feature of the second class, however, is the fact that they always occur behind a vessel so large that the retinal layers behind it, as well as the limiting membrane, are curved outward and the rods and cones much shortened (Fig. 4). The displacement of the nuclei in the second class then seems to be brought about by pressure of blood vessels. It is less likely that it could be due to atrophy resulting from the inability of the cones behind the vessels to carry out their functions.

The external limiting membrane of the retina stains deep blue, the hyaloid membrane reddish-brown. Neuroglia fibres in both the retina and optic nerve are well stained, but are not so sharply differentiated from the nervous elements as by the foregoing method. However, this method has the advantage over the latter of being constant in its results and of preserving neuroglia cells and nuclei more perfectly. In glioma retinæ by this method also the writer has always failed to find anything resembling neuroglia fibres in the tumour proper. But the cells and nuclei of these tumours, the limiting membrane of the rosettes, with the little projections through it (rudimentary rods and cones) are most perfectly brought out.*

Note on the Musculus Papillæ Optici of Nikolaï.

The cells in the optic disc, recently described by Nikolaï† as forming a musculus papillæ optici, are deeply stained by

* Mallory's connective tissue-stain also gives interesting results after Zenker's fluid when applied to the retina. It is particularly useful for demonstrating the blood-vessels, the walls of which are stained bright blue. The inner limbs of the rods and cones stain dark red, the walls of the outer limbs blue, and the contents of the latter, orange. The interstitial substance mentioned above stains blue. The external limiting membrane stains red, the hyaloid membrane intensely blue. Müller's fibres and nerve fibres stain red. All nuclei stain orange.

† Annales D'Oculistique, Nov., 1902, p. 342.

the last-mentioned method, and their structure so well brought out as to render the view that they are smooth muscle fibres improbable. Some of these elements appear in the form of long spindle cells with double contours, having rod-shaped nuclei, and certainly bear a close resemblance to smooth muscle. When they lie entirely in the plane of the section, however, they can be seen to terminate in fibrils longer than those ordinarily forming the extremities of smooth muscle fibres. Mixed in with these cells are to be seen shorter cells which break up into one or more fibrils at each end. In fact there are all gradations to be met with between spindle cells with one fibril at each end to stellate cells (not "spider" cells) with several fibrils attached. Some of the cells have scarcely any cell body, and the fibrils are given off almost at the nucleus. Many of the stellate cells are similar in shape to the cells of myxomatous tissue or to the cells of the iris stroma, but differ from them in that the processes (fibrils) of the cells in the disc do not branch and do not unite with the processes of neighbouring cells. In specimens stained by the ordinary methods, it is difficult to make out the fibrils, or at least their relationship to the cells, and the spindle cells cannot be distinguished from smooth muscle fibres. In any case, many of the fibrils are missed because they pass out of the plane of the section.

The only real evidence advanced by Nikolaï, which goes to show that the spindle cells are smooth muscle fibres, is based on morphology. This seems insufficient when it is considered that the cells in question occur in a structure in which there is no physiological indication for the presence of muscle fibres. Moreover, Nikolaï fails to exclude the possibility, which seems most probable, that they are neuroglia cells. It is true that he applied Weigert's stain and obtained negative results, but he overlooked the fact that by this stain only the fibrils and not the cell bodies of neuroglia are stained. So far as the writer is aware, there is no stain which will clearly differentiate neuroglia cells from smooth muscle fibres.

The stellate cells, as well as the other cells with fibrils attached, of course cannot be regarded as smooth muscle fibres, and since the fibrils of the spindle cells are entirely similar to those belonging to the other cells of the disc, it is reasonable to suppose that all of these cells are of the same nature, and that the spindle cells, too, are not smooth muscle fibres. An additional argument in favour of this view is the fact that the writer has seen, under pathological conditions, marked proliferation of the cells of the disc with the production of many new spindle cells possessing fibrils.

But if the cells and fibrils of the disc, including the spindle cells, are neuroglia elements, they differ from the neuroglia found in the retina and optic nerve proper, and from that in the brain and spinal cord. As is well known, it is a matter of dispute as to whether or not neuroglia fibres in the central nervous system are the processes of cells, Weigert* vigorously upholding the negative side of the question. It has been claimed that the neuroglia fibrils pass through the cells unchanged. In the trunk of the optic nerve, the writer has not been able to convince himself that the fibrils are in continuity with cells, although some of the cells certainly have short processes. It is also difficult to determine whether the fibrils pass through the cells or simply run along their sides. Here, too, one of the arguments, advanced in case of the central nervous system, seems to hold good, namely, that the fibrils are too numerous and the cells too few for each fibril to be connected with a cell. At the lamina cribrosa, however, the character of the neuroglia rather abruptly changes, the fibrils become coarser and much less numerous, and, in fact, seem identical with the fibrils attached to the cells described by Nikolaï. Moreover, the cells in the disc are sufficiently numerous to substantiate the view that each fibril is attached to a cell. The fibrils in the optic disc differ also as regards staining properties from those in the nerve trunk, for after fixation

* Beiträge zur Kenntnis der normalen menschlicher Neuroglia. Frankfurt-a-M., 1895.

in formaldehyde alone or even when followed by nitric acid, they sometimes fail to stain in specimens in which the fibrils of the nerve trunk stain deeply.

On the other hand, the writer has found the same kind of cells and fibrils, including the spindle cells, in a true glioma of the brain, and in cases of phthisis bulbi, such cells were found to make up a large part of the new-formed neuroglia arising from the retina. They also occur in the retina under other pathological conditions. It would seem, therefore, that the spindle cells in the optic disc, as well as the other cells with fibrils attached, are really neuroglia elements, but that they represent a less highly differentiated form of neuroglia than that in the optic nerve proper.* And it seems more than probable that the spindle cells are modified spongioblasts, and are closely related to Müller's fibres. This view is borne out by the fact that in the retina just posterior to the ora serrata, there are frequently to be found spindle cells similar to those in the disc. It is also consistent with the fact that the cells in question occur at the point of transition between the optic nerve and retina.

Description of Plate.

1. Separated retina, showing Müller's fibres. Formaldehyde fixation.

2. Arrow-head markings in medullated nerves. From a case of plexiform neuroma of the eye-lid. Formaldehyde fixation.

3. Proliferation of neuroglia in atrophied retina (gliosis retinæ). Formaldehyde fixation, followed by nitric acid.

4. Normal retina, showing shortened cones behind blood-vessel with nuclei external to membrana limitans externa. There is another vessel on the left. Fixation in Zenker's fluid.

* The question of the occurrence of differentiated and undifferentiated neuroglia fibres in gliomata of the brain has been discussed by E. W. Taylor, Journal of Experimental Medicine, 1897, p. 611.

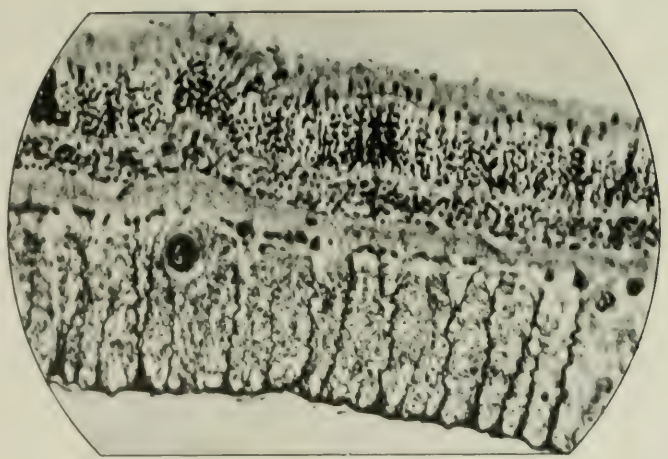

Fig. 1.

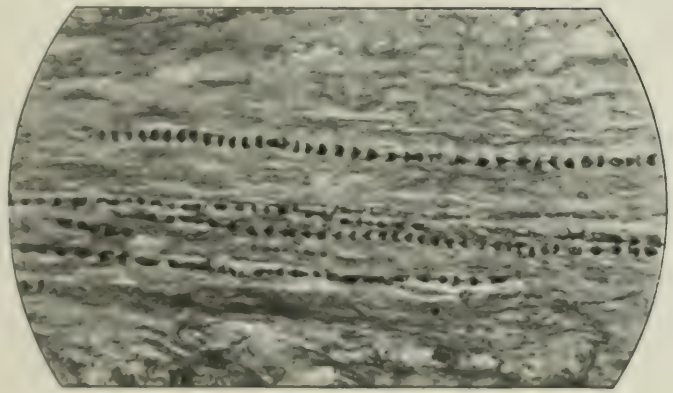

Fig. 2.

Fig. 3.

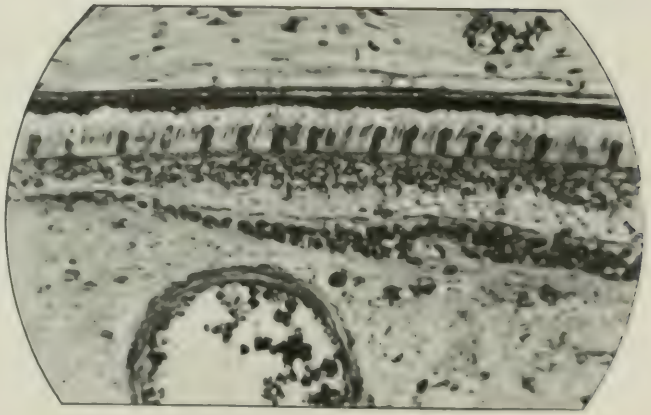

Fig. 4.

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II. A HITHERTO UNDESCRIBED MEMBRANE OF THE EYE, AND ITS SIGNIFICANCE.

WHILE studying the external limiting membrane of the retina in a section of a normal eye, the writer was surprised to find it continue on at the margin of the disc and pass into the layer of pigment epithelium, where it could be followed, until the gradually increasing pigmentation hid it from view. Other sections of the same eye, bleached by the method of Alfieri,* and stained in a variety of ways, showed the still more surprising fact that this membrane was present in the pigment layer throughout the fundus. The eye from which the specimens were obtained was removed from a man aged 65 years, who suffered from extensive carcinoma of the eye-lid with probable involvement of the orbit. It was opened immediately after enucleation and fixed in Zenker's fluid. The writer has since found the membrane in eyes fixed in formaldehyde, but in these the pigment layer is not so well preserved. A few sections from an eye fixed in formaldehyde and containing a glioma retinæ were particularly fortunate. They passed through a portion of the pigment layer in which the pigment was almost entirely absent, and therefore did not require bleaching. Mallory's[2] phosphotungstic acid hæmatoxylin seems to be the best stain for the membrane, because in specimens fixed in formaldehyde, and in bleached specimens fixed in Zenker's fluid, it does not stain the cells to any extent, and the membrane is brought out in sharp contrast. The ordinary alum hæmatoxylin and eosin method of staining, however, gives fairly satisfactory results, the membrane taking the eosin stain.

* Celloidin sections are placed in a solution of permanganate of potash, 1 : 2000, for 24 hours, and then in oxalic acid, 1 : 300, for a few hours. The permanganate solution should be made up fresh each time, and a large volume of it should be used. If the bleaching is not complete, the process can be repeated.[1]

The appearance presented by this membrane in cross sections is that of a delicate line running along near the inner margins of the cells. The latter project beyond it in the form of processes of variable length, showing from this fact alone that the line does not represent the inner contours of the cells. Occurring at almost regular intervals on the line, there are black dots, each of which on close examination is found to be situated at the line of junction of two cells. On careful focusing it can be seen that the line is not always on the same level, evidently passing sometimes behind and sometimes in front of the cells; rarely it is missed entirely for a cell or two. This makes it difficult to obtain a good photograph of the membrane. In oblique sections the true structure of the membrane is at once apparent, the latter appearing then not in the form of a straight line, but in a series of hexagonal loops, which are fused together at their sides of contact so as to form a screen into the openings of which the pigment cells project. The little dots resolve themselves into the sides of the loops, which in cross-section had pointed more or less directly at the observer. Fig. 1 shows the membrane in both cross and oblique section.

That this is a true fenestrated membrane, and is not an appearance produced by the contours of the cells, there can be no doubt whatever. In the first place, as just stated, it does not correspond in position to the margins of the cells. Secondly, in phosphotungstic acid hæmatoxylin followed by acid fuchsin it stains blue, whereas the cells stain pink, or not at all, and show no vertical blue lines between them. Thirdly, in certain places where two neighbouring cells have shrunken apart, it remains intact. Fourthly, it can frequently be seen projecting beyond a fragmented portion of the pigment layer. Such a projection in cross section is shown in the photograph, Fig. 2. The writer has also not infrequently seen, even in unbleached specimens, horizontal loops out of which the cells had fallen, which were still in continuity with the loops of adjoining cells, Fig. 3.

Moreover, this membrane has nothing whatever to do

with the cement substance or neurokeratin envelope that has been described as lying between or covering over the cells of the pigment layer. While there is no reason to doubt that such a substance exists, it does not stain in the specimens of the writer. However, on carefully focusing, a bright line or space can be made out between the cells, which in some instances is especially plain. The reason it cannot always be seen with the same distinctness is probably due to the fact that the cells are hexagonal in shape.

As already mentioned, in phosphotungstic acid hæmatoxylin the membrane stains blue in both bleached and unbleached specimens. In the specimens from the case of glioma retinæ which required no bleaching, the neuroglia of the optic nerve, Müller's fibres of the retina, the rods and cones, and the external limiting membrane, were also stained. In bleached specimens of the normal eye fixed in Zenker's fluid, the only other structures to take the stain were the external limiting membrane, and a large number of neuroglia fibrils in the optic nerve. Neither membrane stained by Weigert's elastic tissue stain. Mallory's connective tissue stain[2] could be applied of course only to unbleached specimens fixed in Zenker's fluid, and in these the visible part of the membrane in the pigment layer and the external limiting membrane were stained a deep red. Staining reactions, therefore, seem to offer strong evidence that both of the membranes consist of a substance identical with or closely allied to neuroglia.

Not only does the membrane of the pigment layer stain in the same way as the membrana limitans externa, but it has also the same structure. The component loops of the latter membrane are of course smaller, since they surround smaller structures, but although the loops for the rods are particularly small, each rod as well as each cone has its individual loop. For this reason the dots seen along the membrane in cross section occur at much more frequent intervals than in the case of the membrane of the pigment layer. The small size of the loops makes it difficult to determine their exact shape, but in general they seem to be round. However,

at a short distance from the optic disc, where the rods and cones assume the form of short thick processes, the loops are unmistakably hexagonal in shape.

Another fact of importance, which indicates that the two membranes are really identical structures, has already been mentioned, namely, that in some places at least they are continuous at the margin of the disc. This can be explained in one or both of two ways. It could be the result of the actual continuity of the pigment layer with the invaginated portion of the optic stalk during early embryonal life, or it could be due to the fusion of the two layers at the disc, and the consequent fusion of the membranes. The specimens of the writer seem to indicate that the former is the correct explanation.

The writer has a few specimens which show a similar membrane in the ependymal epithelium of the brain and spinal cord. Possibly this has already been described by others.

For years the external limiting membrane of the retina has been described as being formed either by the apposition of the outer ends of Müller's fibres or by lateral interlacing offshoots from them. Greeff[3] in his elaborate article on the retina still holds to this view. For a long time also it was thought that in a similar way Müller's fibres formed an internal limiting membrane, but this view has now been generally abandoned. There is of course a membrane, the hyaloid membrane, lining the inner surface of the retina, but it is in no way similar to the membrana limitans externa. It stains slightly in Weigert's elastic tissue stain, and intensely in Mallory's connective tissue stain, and is, therefore, evidently of mesoblastic origin. The basis for the belief that the membrana limitans externa is formed by Müller's fibres seems to rest on the fact that the latter have been seen apparently to end in contact with it. In view of the fact, however, that an entirely similar membrane, and one with which it is directly continuous, exists in the pigment layer where there are no such fibres, the writer feels that this theory also must be abandoned. Moreover, although the writer has carefully studied many

sections in which Müller's fibres were deeply stained by phosphotungstic acid hæmatoxylin, in no instance, at least in the normal fundus of the adult, were they found to end in contact with the membrana limitans externa. The little processes that have been described as arising from the membrane and passing in between the rods and cones so as to isolate them, are evidently nothing but the cut ends of loops seen obliquely.*

No one can doubt that the membrane in the pigment layer is a product of the cells which project through it. It seems clear, therefore, that the membrana limitans externa must have a similar origin. The latter membrane, however, makes its appearance long before the rods and cones are formed, so that it must be manufactured by cells which later become converted into these elements. The natural deduction from these conclusions is that the rods and cones, and the cells of the pigment layer, are essentially of the same nature, and that the former are really only modified spongioblasts (of His). Two facts already mentioned support this deduction, one is that at the margin of the optic disc the rods and cones can sometimes be seen gradually to assume the character of the pigment cells, and the other is that the membrana limitans externa has the characteristic staining reactions of neuroglia. In the preceding article, too, it was pointed out that the rods and cones, and frequently also their nuclei and fibrils, could be differentiated from the bipolar cells of the retina by the neuroglia stain. The theory that the rods and cones represent modified ependymal cells is not new,† but it seems to the writer that the foregoing considerations make this theory practically a certainty. It is interesting that this supports Barker's[5] contention, based on analogy, that the rods

* The appearance of little processes is also sometimes produced by the contours (membranes) of the cones, which are especially distinct near the membrana limitans externa. They are also to be seen running inwards, and may be mistaken for fine branches of Müller's fibres.

† W. Krause[4] regards the rods and cones as ependymal cells with their cilia matted together.

and cones are not nervous elements, but sensory epithelium, and that the bipolar cells of the retina correspond to the cells of spinal ganglia.

As is well known, in the rosettes of glioma retinæ there is a membrane which on cross section appears to be identical with the membrana limitans externa of the retina. So far as the writer can ascertain, however, its appearance in plane section has never been described. To determine the structure of this membrane, a glioma retinæ was selected in which rosettes were both unusually large and numerous. The globe had been opened immediately after enucleation and portions of the tumour fixed in Zenker's fluid, formaldehyde, and alcohol respectively, but Zenker's fluid was found to give by far the most satisfactory results. Sections from the portion of the tumour fixed in this fluid, show conclusively that the membrane of the rosettes is a fenestrated structure similar in every way to the membrana limitans externa and to the membrane of the pigment layer. The same stains were applied as in the case of the other two membranes and with identical results. In these specimens the fenestrated structure can be made out in the great majority of the rosettes, but in some of them it is especially distinct. Fig. 4 shows a rosette in the upper part of which the membrane is cut transversely, while below it is cut in plane section and can be seen to become continuous with a series of loops. It is noteworthy that these loops are always hexagonal and of about the same size.

In the centres of many rosettes there are other hexagonal figures whose outlines are coarser than those of the true membrane. Such figures are also frequently seen elsewhere in the tumour. They can be easily distinguished from the membrane by means of Mallory's connective tissue stain, which stains their walls slightly blue, while the membrane is stained a deep red. This stain seems to show a definite intercellular substance throughout the tumour which takes a blue tint. It does not stain sufficiently blue, however, to indicate that it is a reticulum of connective tissue. The hexagonal figures in the centres of the rosettes, therefore, no doubt

represent the walls of spaces out of which cells have fallen.

The structure of the membrane in the rosettes explains why the latter assume their peculiar form, for it is evident that if a few cells became bound together by such a membrane, and they were otherwise unhampered in their growth, they would be compelled to form a more or less spherical body. If the free ends of the cells groups did not finally meet, a spiral figure would result, and just such spirals are frequently seen. The writer has also found single cells with a membrane on one end, as well as curved groups of several cells which were obviously the beginnings of rosettes. This explains very well why tumour cells are often seen within the cavity of rosettes, evidently being enclosed during the formation of the latter.

The similarity of the rosettes of glioma retinæ to the layer of rods and cones was first pointed out by Flexner[6] and a few years later by Wintersteiner.[7] Although the work of these two observers was unquestionably independent, their conclusions were almost identical. Each took the view that the rosettes indicated a tendency on the part of the tumours to form rods and cones, and each suggested the term neuroëpithelioma as the proper designation for these tumours. Flexner's conclusions were based upon the study of one case only, which he evidently considered to be of a different nature from the usual type of glioma retinæ. He also thought that he could locate its origin in the external nuclear layer of the retina. Wintersteiner,[8] on the other hand, studied a large number of cases, and came to the conclusion that the term neuroëpithelioma was applicable to them all, although most of them did not contain rosettes. The latter observer found also that these tumours probably started in the majority of cases from the internal nuclear layer.

A strong argument against the theory of Flexner and Wintersteiner in regard to the nature of rosettes, is the fact that according to accepted views the membrane of rosettes could not be formed in the same way as the membrana

limitans externa, since there is nothing corresponding to Müller's fibres in these bodies. Strangely enough, this fact seems to have been entirely overlooked. The observations of the writer, however, render this objection no longer valid, for it has just been shown that the membrana limitans externa is in all probability produced by the cells which go to form the rods and cones. It was pointed out, too, that the two membranes are identical in structure.

Sections of the tumour mentioned above, that was fixed in Zenker's fluid, show a closer resemblance between the rods and cones and the corresponding processes of rosettes than is realised. In some rosettes there are conical processes projecting beyond the membrane, the bodies of which stain deeply in phosphotungstic acid hæmatoxylin, and which terminate in unstained extremities. These processes in fact are indistinguishable from some of the thickened cones seen in a normal retina near the the optic disc and in the neighbourhood of the ora serrata. Moreover, the membrana limitans externa near the optic disc, as already noted, is made up of hexagonal loops as in the limiting membrane of rosettes. This shows, of course, that in both cases the cells are hexagonal in shape. It should not be overlooked that it is only here and there that one can expect to obtain a perfect longitudinal section of a rod or cone in spherical bodies like rosettes.

Flexner and Wintersteiner applied the term neuro-ëpithelioma to the tumours under discussion, because they regarded the latter as being composed of elements corresponding to the rods and cones, the neuroëpithelium of the retina. While, however, the latter name, first given by Schwalbe[9] to the rods and cones with their nuclei and fibrils, has met with general acceptance, no entirely satisfactory evidence has been brought forward to show that they are not really nervous elements. The observations of the writer, however, already given, strongly support the view that they are not nervous elements, but only slightly modified spongioblasts.

While then the nature of the rosettes in glioma retinæ seems plain, the character of the other elements in these tumours is still undetermined. Thus far there is no trustworthy evidence that the cells are differentiated into true ganglion cells or neuroglia cells. Greeff[10] claims that by means of the Golgi-Cajal method he has not only demonstrated several types of ganglion cells, but neuroglia cells as well. If ganglion cells can be found in cheese by this method, it is not surprising that they should have been found in a tissue so prone to undergo degeneration as is glioma retinæ. Then, too, the ganglion cells and neuroglia cells belonging to a portion of retina incorporated in the tumour might easily be mistaken for elements of the tumour itself. As stated in the preceding article, the writer has been unable to find neuroglia fibres in these tumours, which were not connected with the retina, by Mallory's method or by any of the modifications of this method there given. Brown Pusey[11] has recently described a case that he evidently regarded as glioma retinæ, and has arrived at the conclusion that glioma retinæ consists essentially of neuroglia elements. He also concluded that the limiting membrane of rosettes corresponded to the internal, not to the external, limiting membrane of the retina. Judging from the description of the case, however, the writer is very sure that it is really one of phthisis bulbi posterior following metastatic ophthalmitis, one of the conditions known clinically as pseudo-glioma. The "rosettes" were evidently nothing more than retinal folds.

In view of the fact, then, that there is no reason to believe that these tumours contain neuroglia, it is obvious that the term glioma retinæ is an inappropriate one. It is all the more so because there are tumours of the brain, termed gliomata, which are believed to be made up of neuroglia, and it seems possible that such tumours may yet be encountered in the retina. The term neuroëpithelioma, however, is no less inappropriate than the other. If it indicated no more than that the tumour took its origin from the epiblast of the neural

canal of the embryo, it would unquestionably be a most suitable term. Unfortunately, the term neuroëpithelium has been applied to the layer of rods and cones in the fully differentiated retina, and "neuroëpithelioma" therefore indicates a tumour whose essential elements correspond to the rods and cones. But, as a matter of fact, it is rare for rosettes to form more than an insignificant part of the tumour, and in most of these tumours they are entirely absent. It is possible, if not probable, that they simply represent a differentiation of the essential elements of the tumour, and that the latter may undergo differentiation in other directions as well. The possibility remains that many of the elements in these tumours are neuroblasts. It is evident, therefore, that "neuroëpithelioma retinæ" is a no more suitable designation than "glioma retinæ" for the tumours to which they have been applied, and that at present there is no reason why the latter should be supplanted by a term over which it has long priority.

Conclusions.

In the pigment layer of the retina there is a fenestrated membrane, identical in structure and staining reactions with membrana limitans externa.

The rods and cones are not nervous elements, but modified ependymal cells, and are analogous to sensory epithelium.

The limiting membrane of the rosettes of the glioma retinæ is a fenestrated membrane similar in every way to the membrana limitans externa, and the rosettes correspond to the neuroëpithelium of the normal retina.

The structure of the limiting membrane of rosettes explains why they assume their spherical and spiral-like forms.

"Neuroëpithelioma retinæ" is no more suitable than "glioma retinæ" for the class of tumours to which these terms have applied.

The writer desires to express his thanks to Mr. J. Herbert Parsons, to whom he is indebted for the microphotographs illustrating this and the previous article.

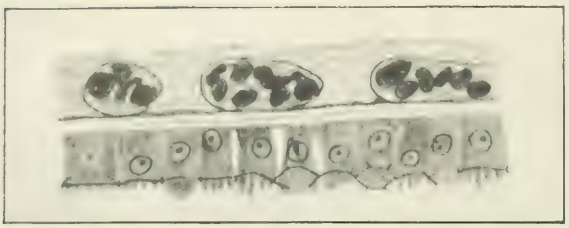

Fig. 1.

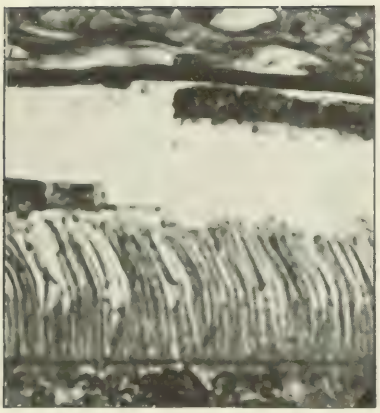

Fig. 2.

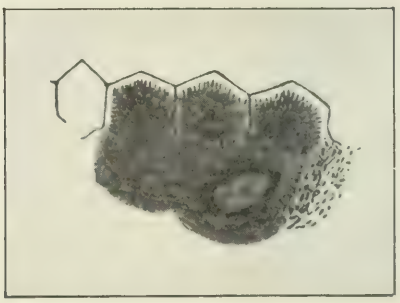

Fig. 3.

Fig. 4.

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(11.) Brown Pusey. The Genesis of Glioma Retinæ in Neuroglia. Johns Hopkins Hospital Bulletin, 1902, p. 229.

Description of Plate.

1. Pigment epithelium with pigment removed, showing fenestrated membrane in both cross and oblique section.

2. Photomicrograph, showing a loop of the membrane in cross section extending beyond a fragmented portion of the pigment epithelium.

3. Isolated group of unbleached pigment cells lying transversely, showing hexagonal loop of membrane partially freed.

4. Rosette of glioma retinæ, showing limiting membrane in cross and plane section.

OBSERVATIONS ON RENAL RETINITIS.

By E. Nettleship.

(The following paper formed the introduction to a discussion on "Eye Changes in Relation to Renal Disease" at the meeting of the British Medical Association at Swansea on July 30, 1903. The paper is based chiefly on my records of all the cases—rather more than 100—that I have seen and noted, and the case numbers refer to these. The parts within rectangular brackets [ ] were either communicated to me by their authors after the paper was written, or formed part of the discussion.—E. N.)

Although much attention has been bestowed upon the retinal changes that occur in renal disease, information is still needed upon several points, from physicians and general practitioners as well as from ophthalmologists, and the following heads are suggested as containing material suitable for discussion:—

1. It is, of course, in the chronic interstitial and chronic parenchymatous forms of nephritis that albuminuric retinitis so commonly occurs. A few cases are recorded in connection with lardaceous disease, and with nephritis due to inflammation of the bladder, ureter, or pelvis of the kidney, but additional cases in connection with any of these maladies are deserving of careful record. The same may be said of retinitis due to acute nephritis of previously healthy kidneys; such cases are said to occur occasionally, especially after scarlet fever.

Eyre* records the case of a man, aged 50, with neuro-retinitis and the characteristic pattern of white dots around the yellow spot, malaise but no distinctive symptoms, and urine, for the first 10 to 14 days of the illness, free from albumin and casts. He died 2 months after the commencement of failure of sight (which was one of the earliest symptoms, and dated only 3 days before the first ophthalmoscopic examination), uræmic and

* Eyre, Ophthalmic Review, vol. xvi, p. 275, 1897.

dropsical, and at the post-mortem the kidneys were swollen and enlarged (together weighing 12·5 ozs.), the capsule non-adherent, cortex swollen and lighter in colour than the pyramids, which were extremely congested. The heart is not mentioned. No doubt in this case there was acute nephritis, but the fact that the casts found in the earlier period are specially stated to have been granular and hyaline, and that no blood was found in the urine till later, seem to indicate that some degree of chronic nephritis had been present before. The absence of any note on the heart, and of a microscopical examination of the kidneys, also lessen the value of this otherwise carefully recorded case.*

In regard to albuminuric retinitis from chronic nephritis due to conditions of bladder, ureter, or pelvis of the kidney, I can mention the following cases:—

(1) A boy (No. 62), aged 15, who had been operated upon for stone when $4\frac{1}{2}$ years old. Dr. Sharkey sent him to me with severe retinitis in February, 1889; the boy was ill and his urine highly albuminous. He was said never to have had scarlet fever. Arteries thickened; accentuated second sound; urine abundant (usually about 60 to 75 ozs. in 24 hours), clear, acid, S.G. 1010, containing albumin in variable, but often large, quantity. Discharged in improved health after 2 months' treatment as in-patient under Dr. Harley.

(2) A man, æt. 49 (No. 78), who for many years had had some degree of cystitis, following Holt's operation for stricture of the urethra performed when he was 35.

(3) An old man (No. 80), whose renal symptoms and consequent retinitis appeared late in life, when he was 76, apparently in connection with enlarged prostate and cystitis.

2. But the chief exception to the rule that the retinal changes depend upon chronic nephritis is seen in the albuminuric retinitis of pregnancy. It appears to be well-established that a peculiar form of kidney disease occurs in

* Dr. C. R. A. Sutton mentions briefly two cases of retinitis during a first attack of acute nephritis, but does not furnish sufficient details. Guy's Hospital Reports, vol. lii (1895).

pregnancy, the special character of which is rapid degeneration of the epithelium, such as is found in some forms of acute blood-poisoning. Though this condition is not a true acute inflammation,* the kidney so affected may pass with unusual rapidity into a state of chronic nephritis and contraction.† Chronic nephritis of the ordinary forms may, of course, be present before the first pregnancy. In connection with our present subject, what we have to ask for particularly is that in every case of pregnancy retinitis, any evidence as to the state of the kidneys before the first pregnancy should be recorded; proof on the one hand that they were diseased, or evidence, based on careful examination, that they were sound. [Mr. Marcus Gunn informs me that in all the cases of albuminuric retinitis of pregnancy that he has seen in recent years, disease of the retinal arteries has been well-marked. This observation is important if the retinitis in any of Mr. Gunn's cases occurred in the first pregnancy.—E. N.] The propriety or otherwise of prematurely emptying the uterus may depend in part upon the conclusion as to the character of the kidney disease, whether old and progessive, or recent and perhaps recoverable.

It is generally held that the prognosis for life in the renal retinitis of pregnancy is better than in the ordinary cases, and my own experience agrees with this. This does not of itself prove that the renal disease of pregnancy is peculiar; it may merely show that pregnancy acts by causing an exacerbation of pre-existing kidney disease, from which exacerbation the patient recovers after removal of the special cause. But examination of the facts does, I think, support the hypothesis that in many cases the kidneys were healthy until they were attacked by the special pregnancy disease. Thus out of 22 cases of pregnancy retinitis, of which I have notes, I know of only five deaths, and only one of these

* Herman, Article: "Eclampsia," Allbutt's System, vol. vii. Groenouw, Graefe-Saemisch, Handbuch, 2nd edn., Kap. xxii, 1901.

† Dickinson, Article: "Diseases of the Kidney," Allbutt's System, vol. iv.

(No. 13) occurred within 2 years of the retinitis. (The four others died respectively $3\frac{1}{4}$, $4\frac{1}{4}$, 7, and 7 years after the retinitis.) Six other cases, of whose death I have no record, are known to have lived more than 2 years (one of them at least 5 years, one 9 years, having had pregnancies subsequent to the one that caused the retinitis; one $5\frac{1}{2}$ years, one 11 years, one 10 years, having had two pregnancies since the first which caused the retinitis; one 13 years, one 24 years). So that of the 22, nine (or two-fifths) are known to have lived 2 years or more after the retinitis, whilst of the four known to have died, only one, as stated above, succumbed in less than 2 years. On the other hand, of 42 non-pregnancy cases in which I know the duration of life after the retinitis, only nine or just over one-fifth lived more than 2 years: and no less than 25 died within 1 year. So it seems that if the pregnancy patients survive the ordinary maximum at all, say 2 years, they are likely to live for several years and sometimes for the full term of life. As a rule, the retinitis of pregnancy occurs only once, but occasionally the history points to a second more or less definite attack.

Again, in the majority of my own cases retinitis has not occurred until after several pregnancies, and in many there was a history of several attacks of pregnancy dropsy, the ages of these patients varying from 29 to 41. In 19 cases where the point is noted, the retinitis occurred during the fifth, or a subsequent pregnancy, in no less than 14, whilst in four of the 19 (Nos. 31, 52, 74, 79) it came with the first pregnancy.

Although in two of these four first-pregnancy cases (Nos. 31 and 79) further pregnancies are known to have occurred, no return of renal or eye symptoms took place, and the patients lived at least 10 years afterwards. These two women were very young (19 and 21 years respectively) at the date of the first pregnancy and of the retinitis, and though one of them (No. 79), now at the age of 31, presents the signs of granular kidney, it seems not improbable that the kidneys in both of them were healthy before the original

pregnancy. In other three cases (Nos. 72, 91, 96), where the retinitis, though not occurring in a first pregnancy, was followed by many years' (at least 11, 13 and 24) freedom from symptoms, it is reasonable to draw the same inference. These five cases (Nos. 31, 79, 72, 91, 96) were briefly as follows :—

Elizabeth B. (No. 31). Seen in July, 1881, æt. 28. In first confinement, 9 years ago, was "blind from dropsy" for 2 weeks. Number of pregnancies since then not stated, but after one of them had three fits, and during two of them dropsy, but less severely than the first time, and with no return of failure of V. Two years ago ulcer of L. cornea, leaving scar and preventing good ophthalmoscopic examination. R. V. 6/18, slightly improved by −1·5 cyl.; pigment spots all over fundus, including y.s.; at y.s. also many round dull whitish dots in or beneath retina; o.d. pale; the pigment spots often rather angular; no atrophy of choroid.

Alice T. (No. 79). Dropsy began about 5th month of first pregnancy when æt. 21. At 6th month (May, 1893) convulsions and general dropsy; labour artificially induced; was unconscious 10 days, and as soon as consciousness returned was quite blind; a few days later Dr. A. B. Batley found o.ds. indistinct, and large hæmorrhages into retinæ; u. contained casts and much albumen. Dropsy quickly disappeared, health improved, and on June 18th, 2 weeks after total blindness was discovered, could see dimly with L., but R. still blind. In October, seen at Moorfields, R. V. fingers 1·5 m., L. barely hand reflex; general retinal haze, some characteristic bright white deposits, much disturbance of pigment epithelium, and some blotches of increased pigment; o.ds. not much altered. U. hyaline casts and considerable albumen. About November V. rapidly improved, and she was able to resume her work in a drapery shop. Three years after first pregnancy Dr. Batley again induced artificial labour for commencing dropsy and albuminuria at 7th month of second pregnancy. Sight not affected. Two years after that, third confinement at full time (æt. 26) without recurrence of symptoms; child did well. May, 1903, now æt. 31, widow 3 years. V. has been as now since

about November, 1893. R. – 3 D. cyl. 6/9 partly, L – 2 D. cyl. with – 1 D. sph. 6/12; much disturbance of epithelial pigment about y.s., and some spots of increased pigment; no white deposits; o.ds. normal colour; arteries not bright, but one in L. eye compresses its companion vein decidedly. Radial arteries, especially L., thick and difficult to compress. Heart, second sound much accentuated, and probably both ventricles hypertrophied (Mr. L. J. Paton); u. 1030, high colour, acid, cloud of albumin to heat, nitric acid and picric acid. Considers herself well, and lives carefully.

Mrs. H. (No. 72). Two pregnancies, with interval of several years; second ended at 8th month, æt. 39, child living, albumin and failure of V. shortly before confinement. 5 to 6 months after failure of V. seen with Dr. A. C. Turner; subsiding characteristic neuro-retinitis of both; V.R. 6/24, L. 6/12. April, 1903, æt. 49, Dr. Turner writes that there have been no more pregnancies and she keeps fairly well, but that V. became slowly worse after I saw her and she is now quite blind of both eyes.

Mrs. R. (No. 91). Seen in 1896 and again in 1899, when æt. 65. Six pregnancies between ages of 29 and 41, and had albuminuria during several of them; all either still-born or miscarriages. About 8th month of No. 6 V. failed in both eyes "from albuminuria;" artificial labour induced; V. improved but not to full recovery. Still has albumin at times and has to diet carefully. V. and ophthalmoscopic changes the same at both her visits to me (1896, when æt. 61, and 1899 when 64); R. 6/18, L. 6/60; changes symmetrical, but more in L.; o.ds. pale, retinal vessels not obviously changed, patches of epithelial denudation, with some increase of pigment, at central region, y.s. invaded in L., but free in R.

Mrs. A. (No. 96), when æt. 36, late in a pregnancy, V. failed in both eyes, continued bad till confinement 6 weeks later, and got much worse a few days after birth of child. About 2 weeks after confinement V. began to improve, but never became good again; when at worst was nearly blind. No note as to albumin at that time; but husband (a doctor) says it is now normal. Seen by me for some recent further failure of V., January, 1897,

æt. 49; V. 6/60 with each, and with H. As. 4 D. corrected just reads up to 3 J.; many old pigment spots and pale spots about y.s., and at periphery, both eyes: in R. some fresh hæmorrhages and a single white patch; o.ds. dirty darkish colour and somewhat hazy; retinal arteries small and very bright.

Whilst on this subject of pregnancy retinitis, certain cases may be mentioned in which the sight fails soon *after* confinement, remains very bad for several weeks, and then returns more or less. We do not know the condition of the fundus during the active period in these cases. When seen some time after recovery, traces of past optic neuritis are found in some of them, but in others no visible change remains, and I have never seen signs of widely spread retinitis.* None of the cases that I have seen gave any history pointing to eclampsia, but on the other hand they are not explained either by flooding, or obvious kidney disease. I think that these cases deserve attention, as perhaps furnishing a link between neuro-retinitis from loss of blood, which, after all, may in a sense be toxic, optic neuritis in puerperal polyneuritis,† uræmic blindness, and the more acute examples of albuminuric retinitis.

I saw an additional and well-marked case of this kind with Mr. Doyne, at Oxford, only a couple of days ago. A woman, æt. 30, lost her sight absolutely a fortnight after her first confinement in June, 1902. Optic neuritis was diagnosed by an ophthalmic surgeon in Manchester. The urine was reported, she says, to be natural. She had no fits, and though the labour was instrumental (at the 8th month), she made a good recovery except for weakness. The blindness came on almost immediately after she got up from bed; she was suckling the baby, and did so till it died at 10 weeks of age. After some weeks she began to see light, and improved a little throughout the winter; latterly the improvement has been more marked, and she can now (July, 1903) read short words of print equal to about six Jaeger, and sees about

* Nettleship, R.L.O.H. Reports, vol. xiii, p. 97 (1891).

† Turney, St. Thomas' Hospital Reports, vol. xxv, 1897. "Polyneuritis in Relation to Gestation and the Puerperium."

4/60. The discs are very white, with some haze along the vessels, the arteries are not bright and there are no signs of past retinitis. The urine, tested on 28th August by Dr. R. M. Wood, is free from albumin, and though the second sound is somewhat accentuated, Dr. Wood finds no proof of cardio-vascular changes.

3. Influence of age and sex in renal retinitis (still omitting pregnancy cases).

Sex. In my own cases (numbering between 80 and 90) I find a large excess of males, viz.:—56 males and 30 females, or 64 per cent. and 36 per cent. Miles Miley, in 51 cases found 38 males and 13 females, or 75 per cent. and 25 per cent.; Schlesinger* in 43 cases, 29 males and 14 females, or 68 per cent. and 32 per cent. These figures, 123 males and 57 females, in all 180 cases, show 68 per cent. males and 32 per cent. females.† These proportions, about 2 males to 1 female, are almost the same as in chronic kidney disease (Dickinson and West),‡ and this is what we should expect.

Age. The age of patients of both sexes, with ordinary albuminuric retinitis, ranges between 30 and 60 in about two-thirds of the cases (54 out of 85 in my series), the most prolific single decade being from 50 to 59 (22 cases out of 85).§

If we include the pregnancy cases, the three decades (30 to 60) show nearly equal numbers, and yield together nearly 75 per cent. of the total (69 out of 107); the most prolific decade for the pregnancy cases being between 30 and 39 (10 out of 22 cases). This fact accords with the statement already made, that usually several pregnancies are required to produce such a renal state as will cause retinitis, but it proves nothing as to the condition of the kidneys prior to the first pregnancy.

* Schlesinger, Graefe-Saemisch, new edition, lief. 26-27, p. 97.

† C. S. Bull, however, found only 55 per cent. males and as much as 45 per cent. females.

‡ Dickinson, Clifford Allbutt's System, vol. iv. Samuel West, Lettsomian Lectures, "On Granular Kidney and Physiological Albuminuria," 1900.

§ Eales found the largest proportion of cases in the decade 55–65.—Birmingham Medical Review, 1880.

It is interesting to observe that so far as my small numbers show, the prospect of life seems to be better when renal retinitis comes on after 55 than before; for of my 10 patients who are known to have lived more than 2 years, no less than 7 were above that age when the retinitis occurred, viz.:—56, 57, 60, 64, 65, 75, 76 years respectively. Curiously enough the other 3 patients, who lived an unusually long time after the retinitis, were very young when it came on viz.:—5½, 21, and 23 years of age respectively.

The relative sex frequency is much the same after the age of 60 as before, 10 patients aged 60 and upwards being male, and 5 female, out of 15.

We are in need of more information as to cases of juvenile renal retinitis. I have seen 10 cases in patients under the age of 21 (Nos. 17, 19, 21, 44, 49, 55, 62, 98, 108, 109), and have found 14 others published in sufficient detail. Of these 24 patients, 10 were males and 14 females, almost the reverse of the rule. Of my own 10 cases, 1 (a boy) had been operated on for stone, 1 (a lad of 20 years) suffered from hereditary syphilitic keratitis, and 3 (a boy of 16 and 2 girls of 19 and 12 years) gave a history of scarlet fever about 9 years previously; in the remainder both scarlet and rheumatic fever were denied. I know the result in 6 of my 10 cases:—4 died within 5 months of the retinitis; one (No. 109) lived for 3½ years, dying of phthisis with amyloid disease of many organs; and one (No. 49) is known to be living and apparently well at the age of 25, nearly 20 years after her eye disease. In regard to the nature and cause of chronic kidney disease in children, it is interesting to note (1) Professor Rose Bradford's statement that the atrophic form of chronic Bright's disease, with small white or mottled kidneys, usually occurs in young subjects and is probably not directly related to granular kidney ("The Lancet," 1903, Vol. II, p. 3); [(2) Dr. George Carpenter's opinion, expressed in the discussion on my paper that evidence is accumulating in favour of chronic renal disease in children being often due to hereditary syphilis; (3) Mr. Hartridge, in

the same discussion, mentioned a case of renal retinitis, now under his notice, in a boy, aged 12 years, who is probably the subject of hereditary syphilis.] The 6 cases above referred to are as follows:—

Mary S. (No. 17). Good health till æt. 18, when she had an illness called "inflammation of bowels," and catamenia ceased for a year. Four months before admission into St. Thomas's Hospital another attack of pain in belly with severe constipation; got well from this, but 2 months ago V. failed and has got worse. On admission, 13th February, 1879, æt. 20, single, ill-grown. Has only p.l. Very severe retinitis of hæmorrhagic type with no white patches, but some patches of more or less complete *choroidal* atrophy, probably due to previous hæmorrhages from choroidal vessels. Died 2 months later, some absorption of retinal hæmorrhages having taken place. U. slightly albuminous at some trials, quite free at others. *Post-mortem.*—Kidneys granular, extremely contracted, tough and pale, cortex much wasted; L. ventricle much hypertrophied (an inch thick); pericardium n.; old R. pleurisy. Pregnancy not explicitly negatived.

Louisa D. (No. 19). Measles and whooping cough in infancy; never scarlet or rheumatic fever. Sight defective for 6 weeks before admission, and 17 days before admission a fit with loss of consciousness for several hours. Admitted at St. Thomas's Hospital, 4th June, 1879, æt. 14, under care of Dr. Stone. R. hemiplegia and paralysis of R. external rectus; systolic bruit; u. considerable albumin, a few blood-cells, no casts. Catamenia began 6 to 8 months ago, but have stopped for last 3 months. Severe characteristic neuro-retinitis with much swelling, and the radiating pattern of white deposits around y.s. 30th, V. much worse; urine contains more blood. August—another fit; extensive detachment of retina in each eye. September 23rd.—Died after convulsions. *Post-mortem.*—Heart, great hypertrophy of L. ventricle; old double pleurisy; kidneys, R. reduced to 1 oz. weight, L. 5 oz., granular, capsule adherent and leaving granular surface; brain, large old hæmorrhage just outside L. lateral ventricle. (This case was published by my then house surgeon, Mr. Davidson, in Trans. Ophth. Soc. I. p. 57., but I have

now added some other facts from my own notes made at St. Thomas's Hospital.)

B. (girl, æt. 11), under Dr. Bristowe's care at St. Thomas's Hospital, 11th July to 5th November, 1879, when she died. There was no *post-mortem*. Admitted for headaches and vomiting. Never had scarlet fever. 17th July I found double neuro-retinitis, suggestive of renal disease. U. 1012, a trace of albumin. By end of August I noted "receding renal retinitis; is now quite blind." 5th November—Died.

Thomas H. B. (No. 108). Healthy at birth; had rickets and at æt. 16 months stopped growing, head enlarged, and he began to pass large quantity of u.; æt. 4 years intelligence began to fail. Scarlet fever æt. about 7. Admitted 6th March, 1880 (Dr. Sharkey and Dr. Bristowe; my ophthalmic notes). Tibiæ bent from old rickets; u. 1010, much albumin, a few blood-cells, no casts, quantity about n.; o.ds. hazy; œdema of feet and hands. 11th.—Decided diffuse neuro-retinitis with grey swelling of retinæ. 17th.—Died. *Post-mortem.*—Heart somewhat dilated on both sides; aortic valves diseased. R. kidney $4\frac{1}{2}$ ozs., small, cystic, no visible healthy structure, capsule adherent; L. kidney absent; no trace of it, its vessels or ureter. Dura-mater thick and adherent to skull, parietal bones thick and heavy, brain looks natural.

Theresa R. (No. 109). August, 1876, came to me at the South London Ophthalmic Hospital. She was æt. 12, but undergrown, and looked only 9, and she had enlarged submaxillary glands; severe neuro-retinitis, with many hæmorrhages in retina and some perhaps in choroid; no typical white patches; opacities L. vitreous. U. cloud of albumin repeatedly found. Some hypertrophy of L. ventricle (Dr. Barlow). First-born of 7, of whom 4 were living. Had "inflammation of lungs" æt. 13 months and mild scarlet fever æt. 4, and soon after that a vague illness in which became very weak for a time. In September, 1876, irido-cyclitis of L., with vascular deposits in angle, began and slowly progressed to blindness and shrinking, and in October, 1879, I excised the eye and found detachment of retina. R. had meanwhile recovered from 20/0 to 20/40 with pale o.d., small arteries and some spots like choroiditis. From 4th February, 1880, she was under Dr.

Harley's care, at St. Thomas's Hospital, till her death on 8th April of that year, at the age of about 15½, with advanced phthisis, albumin having continued in the urine till the end. *Post-mortem.*—Dr. Sharkey found old pericarditis and some hypertrophy of both ventricles; kidneys granular with adherent capsule, R. only 1½ oz., L. 5¼ ozs.; lungs destroyed by very large cavities lined by suppurating membrane, but no tubercle found in them. Intestines showed tubercle and some ulceration of Peyer's patches. Liver much enlarged, smooth; spleen enlarged and "sago-grain." Well-marked amyloid reaction with iodine in liver, spleen, L. kidney, and small intestines.

Beatrice H. (No. 49) had whooping cough and measles in early childhood; no scarlet or rheumatic fever; no diphtheria. Is first-born of four. Father has gout in ankle. When æt. 5½, ill with pains in loins and failure of V.; the late Mr. Haynes Walton saw her for the sight, and is reported to have said she had "Bright's disease;" no fits, but was very ill. V. did not begin to return for 4 months. At Moorfields, December, 1886, æt. 8; very timid: stunted and undergrown, patchy congestion of cheeks, heart's impulse increased and apex in nipple-line, radial artery too hard; u. 1010, cloud of albumin (repeated examinations by Dr. Herringham)—V. fairly good, but not normal. O.ds. very white, arteries shrunken, veins show thick white lines; pigment disturbance and some shining white spots at y.s. Seen again in 1887 (March and August), and 1889 (October), and urine each time of low S.G., and contained a little albumin. For a time continued to have much frontal headache, but in other respects improved and grew bigger and stronger. V. was always 6/36 with each eye. In November, 1891, æt. 13, much stronger and better grown; V. of R. 6/36, L. 6/24; U. 1010, but no albumin (single examination). In June, 1903, Dr. W. J. Drew, of Egham, where the girl formerly lived, kindly wrote that he had enquired and heard that she was grown up and in good health. She must now be 25.

4. We recognise two principal factors in the production of albuminuric or, more correctly, renal retinitis, a morbid state of the blood, and a diseased condition of the walls of the

retinal arteries,* and in most cases we have to reckon with both. The blood-state is the chief cause in the severe acute cases with great œdema and effusion, some of which, *e.g.*, the favourable pregnancy cases, recover both health and sight. Arterial disease takes the more important share in many of the less violent cases. Then, we meet with arterial disease, hæmorrhages and retinitis, and white patches, without albumin; and finally cases of arterial disease only—hyaline thickening of coats, with or without exaggeration of the light streak—occasionally with albumin, but more often without.

I do not propose to dwell on the blood-state in renal retinitis. But there is plenty of room for careful clinical work in regard to the diagnostic significance of visible changes in the coats of the retinal arteries.

When we see the retinal arteries thickened and hyaline looking, whether they have caused venous hæmorrhage by compression or not, are we justified in inferring the presence of chronic nephritis if the urine (examined repeatedly) is free from albumin, not of very low specific gravity, and the heart not demonstrably hypertrophied? Dr. Samuel West† believes the diagnosis of granular kidney is justified when we find high pulse-tension, with thickened artery and retinitis, even without albuminuria, and he appears to think that the pulse-tension and thickened artery alone are enough in some cases without either retinitis or albuminuria. The ophthalmic observer may, therefore, fairly suspect an early stage of granular kidney disease whenever he diagnoses decided hyaline thickening of the retinal arteries. The suspicion will be much strengthened if the patient be comparatively young. It is well, therefore, to look for the appearance of such arterial disease by routine in *young* subjects.

This hyaline-fibroid thickening of the retinal arteries is now well-known to be indicated by intensification of the bright streak and compression of the veins where they are

* Gowers, 1876; Brailey and Edmunds, 1881; Marcus Gunn, 1892 and 1898; Hertel, Heidelberg Congress, 1900.

† Samuel West, *loc. cit.*, p. 37.

crossed by arteries. The condition, though probably seldom confined to the arteries of the retina, is, doubtless, often spread unequally throughout the system; indeed, in regard to the retina alone the appearances may be conspicuous in one eye, and insignificant or even absent in the other, whilst even in the same eye they are by no means always equally distributed along the affected vessel or vessels.

I have not myself had the opportunity of tracing a case in which uncomplicated disease of retinal arteries was proved to be followed after some years by the signs of chronic nephritis. Mr. Marcus Gunn,* has however, recorded a very important instance in which a woman, at the age of 51, had bright retinal arteries, and a little hæmorrhage into the vitreous of one eye, but no albumin and no renal symptoms; when 56, there were retinal hæmorrhages and albumin was sometimes present; from 58 to 59 more retinal hæmorrhage, retinal patches, a cerebral seizure, and albumin more frequent; at 61 retinitis worse, cerebral degeneration, uræmic symptoms. I anticipate that other cases of this kind followed from beginning to end will be forthcoming.

[Mr. Marcus Gunn kindly allows me to add the following unpublished cases and remarks, showing, as he believes, that marked arterial disease with albuminuria may sometimes come on comparatively quickly. (1) The patient, a man who had been seen several times during about 10 years by Mr. Gunn, quickly developed serious bad health with albuminuria, degeneration of retinal arteries and hæmorrhages, after a very bad attack of influenza; he died 18 months later of intracranial hæmorrhage. (2) In another patient, whose health suffered greatly after severe influenza, albuminuria, degenerated retinal arteries, and retinal hæmorrhages were found.

Mr. Gunn suggests a comparison between such post-influenzal cases and the albuminuric cases of pregnancy, the arterial and renal disease in both being, he supposes, due to a comparatively rapid poisoning, but the process being usually less severe when caused by pregnancy than by influenza.]

* Gunn, Trans. Ophth. Soc., vol. xviii, p. 356, 1898. (Case 10.)

Whether the thickening of retinal arteries specially described by Gunn and others, is so useful a diagnostic sign of granular kidney when seen in elderly persons, is, I think, an open question. For instance, in my own notes on this point, either the silver-wire appearance, or marked compression of veins by arteries, in patients over 40, has been recorded much more often in women than men. My cases (35 in number), were, however, all in private practice, and may contain some source of error, such as the greater total number of female than of male patients. Mr. Marcus Gunn tells me that he believe she also has seen more women than men with degenerated retinal arteries. In his published series of cases* from the Queen Square Hospital, however omitting such as had albuminuric retinitis, there were 12 males to 9 females, which is more like the normal proportion in chronic renal disease. But even if hyaline thickening of the retinal arteries after middle age should prove to be a less valuable sign of incipient chronic nephritis than it probably is in younger subjects, we must still take it as a sign of the danger of cerebral hæmorrhage.†

5. The general opinion is that the particular characters of the retinitis do not afford any guide to the kind of chronic kidney disease present in a given patient. Dr. Samuel West, however, maintains that the so-called "exudative," or "inflammatory" form of retinitis, *i.e.*, retinitis with much œdema and general haze, indicates parenchymatous nephritis, whilst the "degenerative" variety, *e.g.*, a case characterised

* Gunn, *loc. cit.*

† E. Hertel (*loc. cit.*), who has examined the retinal vessels in a series of patients with widely spread angio-sclerosis, divides the microscopical changes in the arteries and veins of the retina into two groups, those associated with age (senile changes) and those due to inflammation. In the former all the layers show an increase of elastic tissue, and the lumen of the vessel is enlarged, these changes affecting the whole course of the vessels. In the latter the alteration, often limited to certain parts of a vessel, appears in the form of localised thickenings of the intima, which may cause narrowing of the lumen, and are, as in the senile form, also composed of newly-formed elastic tissue.

by the presence of the well-known figure of bright white dots or patches around the yellow-spot, with little or no general haze, but often with decided thickening of the retinal arteries, points to granular kidney. I agree with Dr. West, that the more violent cases of retinitis are chiefly toxic, and the milder, more chronic ones, often chiefly vascular in origin. But I think it is unnatural to draw sharp distinctions between the "exudative" or "inflammatory" and the "degenerative" varieties, or to associate either form with any particular kind of chronic nephritis. If we watch cases from an early stage to the end, we shall find that both in interstitial and in chronic parnechymatous nephritis, the retinitis may be either intense or comparatively mild.

No. 19, already quoted (p. 329), is an example of very severe retinitis, with all the changes in advanced granular kidney disease. In No. 18, a man, æt. 31, had, when I saw him on 3rd October, 1879, diffuse haze and œdema of o.ds. and retinæ without either hæmorrhages or white patches of any kind. A month later (November 5th) o.d. and retinæ clearing, bright white dots at y.s. in R., none in L. Three weeks after this (November 26th), retinæ nearly clear of diffuse haze, radiating bright dots at y.s. very striking; some pigment spots now visible; some hæmorrhages now present. He lived till March, 1880; the kidneys weighed $14\frac{1}{2}$ ozs., capsule non-adherent, surface smooth and generally pale. Heart much hypertrophied.

If the eyes in No. 18 had been examined for the first time in November, the retinitis would, I suppose, have been called "degenerative," and it seems never to have been decidedly "exudative," yet he had "large white" kidneys.

To my mind it is clear that there is only one sort of renal retinitis, and that, apart from arterial disease, the many varieties of change seen during life indicate only different stages and degrees of (*a*) general œdema,* (*b*) exuda-

* Distension of the peri-vascular lymph-spaces of the retina is, in addition to general œdema, described by Weeks, Arch. of Ophthalmology, vol. xvii, p. 276, 1888.

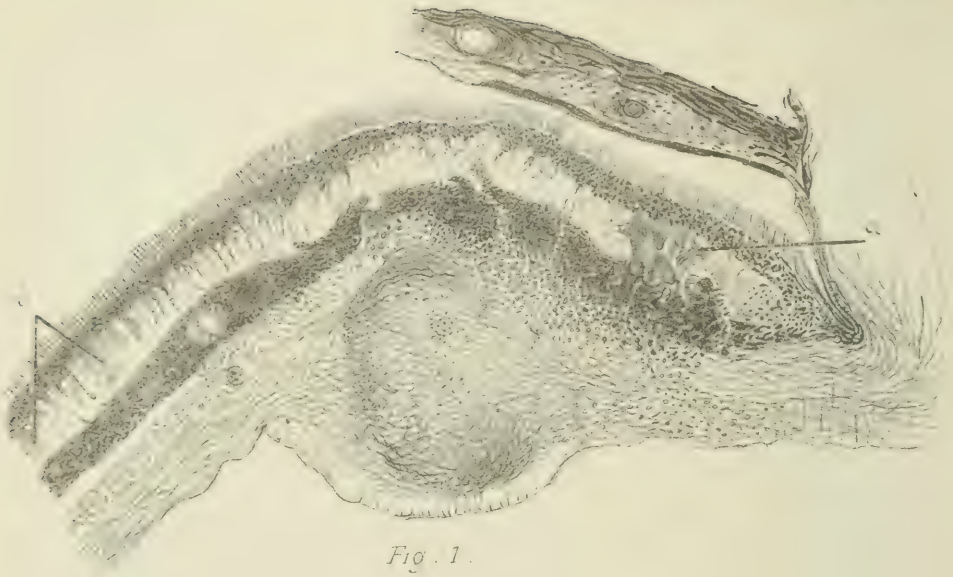

Fig. 1.

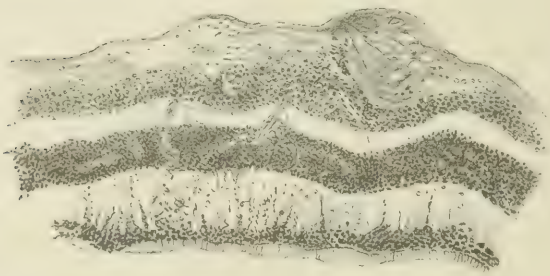

Fig. 2.

Fig. 4.

Fig. 3.

To face p. 336.

Description of Plate.

Illustrating Mr. Nettleship's Paper on Renal Retinitis.

Fig. 1. Shows a large swelling in the nerve-fibre layer of the retina close to the optic disc. There are also amorphous (fibrinous) deposits (*a*) and patches of partly disintegrated blood-corpuscles (*b*) in the inter-granule layer. The letters O D are in the situation of the physiological pit.

Fig. 2.—A smaller swelling in the nerve-fibre layer.

Fig. 3.—Fibrinous deposits in the inter-granule and outer-granule layers.

Fig. 4.—Similar deposits more highly magnified.

The above figures are from sections of the retina of a man æt. 43 (No. 25), in whom Dr. Sharkey found albuminuric retinitis in February, 1880, and who died 3 months later. A few days before death I examined the eyes again and found abundant changes still present. They show that, as stated on pages 336 and 337, in albuminuric retinitis of long standing, the soft-edged patches in the nerve-fibre layer and the sharply-defined deposits in the deeper layers may be present at the same time.

(Figs. 1 and 2 from drawings by the author. Figs. 3 and 4 from micro-photographs by Mr. J. Herbert Parsons.)

Thus in No. 30, a man, æt. 29, had in July retinitis with slight œdema and haze, many soft-edged or woolly white patches, but no bright dots, though these were very carefully sought for. In August (1 month) woolly patches less numerous, but general haze increased, and characteristic bright white dots now present at each y.s. In October (4 months) retinæ had cleared, o.ds. greyish and nearly clear, bright dots still present at y.s. He had on admission signs of parenchymatous nephritis, general dropsy, scanty urine with much albumin and granular and hyaline casts, dyspnœa and some cardiac hypertrophy; he was much better after 6 months' treatment.

The greater the early haze and œdema, the larger is the amount of these intensely white, late, degeneration deposits.* When they appear, the earlier soft-edged patches have often to a large extent cleared off; they last much longer than the soft-edged patches, but if the patient lives they certainly lessen (Cases 44 and 30), and probably disappear in the course of many months, though not in a shorter time. In severe and prolonged cases the different forms of opacity are often so abundant, and so mingled with hæmorrhages and general haziness and discolouration of the retina, that no analysis is possible, and I think that many of the discrepancies noticeable in the published descriptions of renal retinitis have arisen from choosing for illustration cases of great severity and long standing, or cases in which the duration of the changes was not recorded.

6. It is well known that various other constitutional or local conditions produce retinitis or neuro-retinitis resembling, and sometimes indistinguishable from, that caused by disease of the kidneys. The only variety that I think we need dwell upon, however, is the retinitis associated with glycosuria. Retinitis in patients with diabetes is fairly

* The extreme brilliancy of these spots, as seen in ophthalmoscopic examination, suggests that they are of fatty composition; but microscopical examination shows them to be either amorphous or composed of a fine fibrinous network. I have found that neither ether nor chloroform affects their appearance in sections.

common, and, in my experience, is much more frequent in private than in hospital practice.* Thus, of my 59 cases, only seven were in hospital patients. In many the urine contains a little albumin as well as sugar, and in these it may be that we are sometimes dealing with ordinary albuminuric retinitis due to the nephritis that occurs late in diabetes. But I have seen at least a dozen in which there was no albumin, and cases have been published in this country by Stephen Mackenzie,† by the late James Anderson,‡ by Juler§ and by Rockliffe,‖ and in Germany by Hirschberg.¶ I have also recorded cases.** There can, therefore, be no doubt that retinitis may be caused by diabetes. Diabetic retinitis has a strong family likeness to renal retinitis, but differs from it in the absence of the soft-edged or woolly patches, and of the stellate or radial arrangement of the white deposits around the yellow spot, and there is usually no œdema. The hæmorrhages so often present in diabetic retinitis are more often round and deeply seated than linear and superficial, as in renal retinitis; in diabetes they are often derived from capillary aneurysms, not from the rupture of larger vessels in the nerve-fibre layer. The white deposits take the form of irregular masses or clumps rather than dots or round patches, and are often arranged in a rude zone or ring at the circumference of the macular area. In a few instances hæmorrhages into retina and vitreous form the chief feature, and these are, as would be expected, not characteristic of diabetes.

I have the impression that the life prospect in the retinitis of diabetes is better than in ordinary renal retinitis, but my data are too small to justify a positive statement. However,

* Hirschberg makes a similar statement. Centralblatt, 1886, p. 193.

† Mackenzie, R. L. O. H. Reports, vol. ix, 1877.

‡ Anderson, Ophth. Review, vol. viii, 1889.

§ Juler, Trans. Ophth. Soc., vol. xii, 1892.

‖ Rockliffe, *ibid.*, vol. xiv, 1894.

¶ Hirschberg, Centralblatt, 1891, pp. 18, 68.

** Nettleship, Trans. Ophth. Soc., vol. ii (1882), and vol. 6 (1886), with ophthalmoscopic drawing.

this may be, there is no doubt that the age of the patients is generally greater in the diabetic cases. Of 82 patients with retinitis in diabetes (59 of my own and 23 published), only 1 was as young as 35, and the great majority (66) were between 50 and 70. Whilst of 107 with albuminuric retinitis 29 were 35 or less; 46 between 36 and 49; only 32 between 50 and 70 or upwards. Of the diabetic cases 65 per cent. were men, 35 per cent. women, about the same proportion as for the renal cases, and not very different from the respective deaths of male and female diabetics of all ages, which, according to Saundby,* are about 57 males to 43 females.

I have already mentioned that in renal retinitis the prospect for life seems to be better in old persons than in those below 50. If the life prospect in diabetic retinitis is better than in the average renal cases, this may be because the diabetic patients are usually older when the retinitis comes on.

7. Many other points upon which further clinical or pathological information is desirable might be mentioned. I find very little in my notes about either scarlet fever or inherited syphilis as causes of the chronic renal disease leading to retinitis; I hope others who have made more careful enquiries can tell us something.

The typical retinitis of renal disease is of course nearly always symmetrical, though sometimes very unequally so. I have seen several cases of chronic renal disease with changes confined to one eye; in most of these the retinal disease consisted chiefly or entirely of hæmorrhage, the arteries being much diseased. But in one fairly typical case of retinitis (No. 76), the second eye was not attacked till several months after the first.

In another case (No. 59), a man of 48 had rheumatic fever (5 weeks in bed), and 5 months later lost the sight of his L. eye instantaneously from embolism or thrombosis of the retinal artery; soon after this he had an attack of temporary aphasia.

* Saundby, Allbutt's System, III, p. 198.

and then L. hemiplegia, and he was now told that his urine contained albumin: then the R. eye failed with typical albuminuric retinitis, but the L. (embolised eye) in which the arteries were extremely reduced, the disc pale, and V. only 20 J., showed no recent changes at all.

From this case we may probably infer that a retina atrophied by previous disease is unable to take on the œdema and other changes of renal retinitis. Case 64, given below (p. 341), probably illustrates the same point. In another case (No. 22) the patient's L. eye was highly myopic, and showed only two small hæmorrhages and a little mottling of doubtful significance at the yellow-spot; the R., with nearly normal refraction, had typical well marked retinitis.

Knies* mentions having seen "pure hæmorrhagic retinitis" in the hitherto healthy eye of an albuminuric patient, whose other eye had been blinded a year before by retro-bulbar neuritis: the retinitis spared the blind eye.

[Dr. Reeve, of Toronto, related a case of albuminuric retinitis in a patient with myopia 18 D., stating particularly that the retinal changes due to the albuminuria were comparatively slight, though the myopic changes in the choroid were pronounced.]

In this connection it would be interesting to know of instances in which renal retinitis has recurred in the same eye after the first attack had cleared off. I have notes of several cases in which it is almost certain from the history that more than one distinct attack did take place, but I have never actually seen two successive attacks. The following appear to be examples:—

No. 9, a man of 28. His sight failed in June, 1877, when in hospital for kidney disease under the care of Dr. George Johnson and Mr. Soelberg Wells. V. improved and he believes got perfect, but 6 months later (December), renal symptoms returned and sight became worse. In January, 1878, I found (*a*) severe albuminuric retinitis with much swelling, and with detachment of the

* Knies, The Eye in General Diseases, p. 306 (American Translation, 1895).

lower part of each retina; (*b*) many very black pigment spots, some of which, especially at the periphery, were in front of retinal vessels, and also some sharply defined areas "like brown paint," on which the normal epithelial pigmentation appeared to be intensified without being disturbed. The changes under (*b*) were probably left by an attack of retinitis in June, 1877.

In Case 29 the patient, male, 49, had typical retinitis in both eyes when I saw him in May, 1881, and, in addition, at the periphery of L. fundus only, spots of pigment surrounded by a pale zone and evidently old. This eye had been dim for a time in the previous autumn and had recovered, the R. not being noticeably affected at that time.

In Case 64, a woman had dropsy without failure of V. during her third and fourth pregnancies. In the 7th pregnancy dropsy again, and this time V. failed at about 4th month; artificial labour at 7th month. When æt. 29, 5 months after onset of bad sight, Mr. Lawford, in May, 1889, found R. 6 60, L. 6 18, with pale hazy discs, small arteries, bright dots at Y.S. region and pigment spots at equator. Became pregnant again about August, and was confined at full time (8th child) in April, 1890; she said that sight had "failed again" during this pregnancy, and that shortly before confinement she became totally blind. I saw her the following July, æt. 29, quite blind; all the white dots had gone, and only some epithelial changes remained at the Y.S., with the O.Ds. extremely white, and the pigment dots as noted by Mr. Lawford. The appearance suggested additional degeneration of the optic nerves rather than a recurrence of retinitis.

Case No. 50 was that of a woman pregnant 7 times in 7 years. Dropsy during second pregnancy; failure of sight, lasting for 6 weeks each time, during the fifth pregnancy 2 years ago, the sixth 6 months ago, and the seventh 2 months ago. It was for the last attack that she sought advice. All the children premature or stillborn except the second. When seen for third failure of V., January, 1887, æt. 32, typical renal retinitis with V. 6 18. No proof of any old changes, though the history points strongly to previous attacks.

Whether or not a relapse of true retinitis can occur, as I believe it may when no high degree of atrophy follows

the first attack, we have proof that recurrences of retinal haemorrhage do happen in eyes that have passed through albuminuric retinitis; this was seen in Nos. 6 and 96 for example (see below).

In examining cases of albuminuric retinitis, we often find spots of black pigmentation sometimes surrounded by a pale ring; they, no doubt, represent little heaps of retinal epithelial cells, increased by proliferation* and the subsequent contraction of inter-cellular effusion. These pigment spots are generally scattered irregularly, but sometimes are specially abundant towards the periphery, a puzzling fact, since as they are a consequence of the retinal œdema and exudation, we should expect them to be most marked where the retina is thickest and most vascular.

In a few cases actual pigmentation of the retina is observed, as in Case 9, given above, and in the following:—

In Case 83, a woman, aged between 50 and 60, seen in 1894, had, in addition to hæmorrhages and bright white deposits at the Y.S. region, large areas of pigmentation, exactly like retinitis pigmentosa, but confined to the lower part of the equator in each eye. Dr. Knox Shaw, who has seen her more recently, writes to me that the hæmorrhages have recurred during the last few years, and that the retinal arteries have now become extremely small. Though the pigmentation was characteristic when I first saw her in 1894, she did not admit that her sight was worse at night.

In another (No. 96) a presumably eclamptic retinitis occurred quite late in pregnancy, seems to have become worse for a few days after the confinement, and then rapidly improved, V. remaining serviceable. When I saw her 13 years afterwards there were many pigment spots and pale spots both at the Y.S. and periphery, with small, very bright arteries and dirty haze of O.Ds.; vitreous clear. The changes might have passed for old syphilitic choroido-retinitis, but the history was much more like albuminuric retinitis, and, moreover, there were some

* Nettleship. Transactions of Ophthalmological Society, vol. 19, p. 63, 1899.

fresh hæmorrhages in one eye, which were the cause of her coming for advice.

Albuminuric choroiditis is mentioned by many writers, chiefly on the authority of Magnus's monograph, published in 1873.* Magnus could only bring forward a single case, that of an anæmic boy of 15 with albuminuria, casts and mitral stenosis, in whose L. eye a large patch of exudation extended from the O.D. to the Y.S., situated entirely beneath the retinal vessels. Judging from the description and plate, this patch resembled the solitary choroidal exudations with which we are all familiar and which appear to be sometimes tubercular, sometimes gummatous. Neither tubercle nor hereditary syphilis are mentioned in Magnus's account. The exudation disappeared completely in 3 weeks, and no changes were present in the other eye; 3 months later (4 months from the first examination) the renal symptoms were worse, but no changes present in either eye. Magnus also counted as choroiditis the pigment spots which were figured by Liebreich,† and also some cases resembling retinitis pigmentosa, described by Galezowski as "choroiditis nephritica." Notwithstanding the statements of Carl‡ and Knies§ that inflammatory foci are not uncommonly found in the chorio-capillaris in renal cases, I think that such pigmentary ophthalmoscopic changes, as have been just mentioned, should be considered as secondary to the retinitis,‖ or if choroidal at all, as caused indirectly by slow atrophy of the choroid from hyaline-fibroid thickening of its arteries. That the vessels of the choroid are often extensively diseased is well

* Magnus, Die Albuminurie in ihre Ophthalmoskop: Erscheiningen, 1873, Fig. 8A.

† Liebreich, Atlas, Pl. XI, Figs. 1 and 2.

‡ Carl, Hertzog, describes obliterative changes in choroidal arteries and capillaries leading to secondary changes of the pigment epithelium; also definite thickenings of choroid from cellular infiltrations.

§ Knies, *loc. cit.*, pp. 302 and 306.

‖ Nettleship, Trans. Ophth. Soc., *loc. cit.*

known: Weeks has found extravasations of blood into the choroid: and I have also found them in sections from one of my own cases.

Very marked night blindness is noticed occasionally.

One such case (No. 14) was seen in an alcoholic woman, aged 42, with atrophic changes 8 months after severe renal neuro-retenitis; there was no pigmentation of retina, but the retinal arteries were shrunken and one of them ensheathed by white opacity (peri-arterial inflammation). Another case (No. 33) was in a soldier invalided from India for "Bright's disease" at the age of 33. Two years later, in the early summer of 1883, he became rapidly night-blind, and we found contracted fields, pale discs, small retinal arteries and slight haze, but no actual proof of previous gross retinitis; V. from 6/12 to 6/24 according to the illumination. Urine, S.G. 1005, with much albumin. He had been out of work and badly fed for 3 to 4 months when the night blindness came on. In a third case (No. 67), similar to the last, a cabman, æt. 57, who had been underfed and sleeping badly for several months, found his sight failing rapidly at night in the spring of 1890, and we found his sight much worse for Bjerrum's gray types than for black and white. The retinæ were slightly hazy, and showed many small hæmorrhages close to the arteries, but no white patches of either kind were present, and none appeared during the 3 months he was under notice. Urine, S.G. 1010, a considerable quantity of albumin; pulse tension high.

I suggest that in each of these three cases the night blindness was functional, due to exhaustion by bright light in a badly nourished person, whose retinæ were already weakened by arterial shrinking and some degree of previous retinitis.

Mention has been made of white peri-vascular thickening in one of the cases referred to at an earlier part of this paper. This appearance is peculiar: it is quite different from the ordinary opacity bordering the blood column of the vessels near the disc so commonly seen in post-papillitic atrophy, from concentric opaque thickening with contraction

of the lumen, and from the bright "silver wire" appearance described by Gunn. The vessel, which may be either artery or vein, has its blood column entirely hidden for a longer or shorter distance by what looks like a coating of white opacity laid on outside the vascular walls; this coating begins and ends abruptly, the blood column disappearing and re-appearing without obvious alteration of size; sometimes there are little holes or gaps in the white sheath.* A good illustration is given in Fig. 1, Plate XII, of Gowers' Manual and Atlas. I remember seeing the patient from whom that drawing was made, and I have made notes of a good many cases since. It is probably a similar condition that is depicted by Frost† in his Atlas. Mules‡ has also written on the condition and given a coloured illustration. The appearance is not seen until the retinitis has lasted some time. I do not know whether microscopical examinations have been made.

Occasionally new blood vessels form in the vitreous humour. This rare change is, however, not characteristic of kidney disease; indeed, I believe that most of the examples have been seen in cases of diabetic and syphilitic retinitis. I have notes of it in only one renal case (No. 69). Mr. Frost gives one from a woman, æt. 54, with slight albuminuria, but does not mention sugar.§

Iritis in renal disease has been seen in two cases by Knies,‖ but he gives no particulars. Slowly destructive irido-cyclitis occurred in one eye in my Case 109 recorded above, but the inflammation in this eye was probably like that in the intestine and lungs, tubercular and independent of the kidney disease.

* An extremely accurate description of the ophthalmoscopic appearances of this peri-arteritis was given by Hulke in 1865, in an albuminuric case (R.L.O.H. Reports, vol. v, p. 25).

† Frost, Atlas, Pl. XL, Fig. 90.

‡ Mules, Trans. Ophth. Soc., vol. ii, p. 47.

§ Frost, *loc. cit.*, Fig. 91.

‖ Knies, *loc. cit.*, p. 306.

EPITHELIAL PLAQUES OF THE CONJUNCTIVA.

W. T. Lister, F.R.C.S., and W. Ilbert Hancock, F.R.C.S.

We bring forward the following cases of epithelial tumours of the conjunctiva, because in our experience they are very rare, and we think that their classification, ætiology, and certain points in their diagnosis are deserving of careful attention, and also are in need of further elucidation.

Case I.

Martin A., age 24, attended at the R.L.O.H., Jan., 1903, complaining of a small lump on the L. eyeball beneath the upper lid. He had noticed it for at least 10 years, but could not say if it had been present longer. He thought that in the last few years it had slightly increased in size, and he complained that at times *flakes* were detached from its surface by the friction of the upper lid, which, acting as foreign bodies, caused considerable irritation. These flakes he had himself removed on more than one occasion from the lower fornix.

On examination, an elongated oval growth was found in the bulbar conjunctiva, 6 mm. long, 2 mm. wide, situated above the cornea, but separated from the limbus by about 2 mm. The growth was slightly raised, about $\frac{1}{2}$ mm.; it had a sharply defined edge, and was freely movable over the underlying sclerotic.

The surrounding conjunctival vessels were somewhat injected, but the growth itself was *avascular*. Its colour was pale yellow and its surface dull, strikingly different from the normal bright conjunctiva. With focal illumination the surface was seen to be marked by parallel furrows running transversely to the long axis of the growth, a few less distinct furrows were seen running at right angles, *i.e.*, longitudinally with the growth. The lids were normal, there was no history of traumatism, there was no night blindness, and in every other way the eye was normal.

The growth was removed with a portion of surrounding conjunctiva and microscopic sections were made.

Case II.

Mildred S., æt. 15, came to the R.L.O.H., April, 1902, complaining of a small growth on the R. eyeball. Her parents had first noticed it 3 months previously, and could not say if it had been present before that time.

The growth was nearly circular, about 2 mm. in diameter, situated near the limbus, upwards and slightly outwards, for the most part in the conjunctiva, but encroaching about ½ mm. on the cornea. The conjunctival portion was freely movable on the sclerotic. It was sharply defined, about ½ mm. in thickness, of a pale yellow colour, dull granular surface, but in this case no furrows were seen. The surrounding conjunctival vessels were slightly injected. The growth was removed under cocain, but the patient returned a year later with a recurrence exactly similar in position and character. It was removed a second time and microscopic sections were made.

The lids were normal, there was no history of traumatism, no night blindness, and the eye was normal in every other way.

On Microscopic Examination the specimens removed from these 2 cases are found to be so similar that they may be described together.

In each the growth consists of a greatly thickened epithelial layer, which at the margin abruptly passes into that of the normal conjunctiva. The surface cells are squamous and somewhat horny; beneath these are several layers of cells resembling the "stratum granulosum" of the skin, showing well-marked granules which stain deeply. The deeper layers show distinct prickle cells; while in the deepest layers the cells resemble those of the normal conjunctival epithelium.

In the specimen from Case I, in which furrows were seen clinically, some fairly distinct papillæ are clearly visible. In the specimen from Case II, where no furrows were seen, no signs of papillæ are visible.

The subjacent connective tissue shows a patchy infiltration with small round cells, and the vessels are dilated; but in no

part does the epithelium pass down into the underlying connective tissue.

Case III.

J. H., a signal fitter, æt. 51. Three weeks before his visit to the Central London Ophthalmic Hospital, in April, 1899, he had been struck in the R. eye with a piece of stone. He said that no portion of the stone remained in his eye.

He had a small, white, nearly circular, sharply circumscribed growth on the conjunctiva on the inner side of the R. cornea, 3 mm. in diameter, raised about 1 mm., separated 1 mm. from the limbus. The blood vessels round the growth were dilated. The mass was dead-white in colour, with a few minute pink spots on the surface. It was freely movable on the sclera beneath.

The growth was completely removed with a portion of the conjunctiva surrounding it, and microscopic sections were made.

No foreign body was found in the growth, and the eye was otherwise normal.

Microscopic examination of the sections shows the growth to consist of a mass of epithelial cells. In the centre this is six to seven times thicker than the normal conjunctival epithelial layer, while at the margins it gradually becomes thinner. In this case there is no abrupt margin to the growth. The superficial layer consists of squamous cells which are becoming horny, and from this layer one or two processes of horny cells pass from the surface into the mass of epithelium.

Running through the mass a few dilated blood vessels with extremely thin walls are to be seen, and the pink spots seen macroscopically were evidently due to these vessels where they appeared on the surface. There are no definite fibrous tissue processes passing into the epithelial mass; nor do any epithelial processes pass into the subjacent tissue.

At the base of the growth there is some infiltration of round cells.

Case IV.

A case very similar to the last came up at about the same time to the R.L.O.H. We regret that we cannot find the notes of this case nor the microscopic sections, but the case is distinctly

remembered. An elderly patient came to the hosptial with a circumscribed raised tumour on the ocular conjunctiva on the inner side of the cornea, but a little distance from the limbus. It was dead-white in colour, it had no pink spots, as in Case III, and was freely movable on the underlying sclerotic. The microscopic sections showed the growth to be nothing but a mass of epithelial cells.

Comparing these four cases:—

Cases I and II were young people, while Cases III and IV were well over middle life.

In Case III there was a history of injury, and it was after this that the growth was noticed. We think the injury was very likely the cause of the growth, some irritation being set up which caused hypertrophy of the epithelium.

In Cases I and II there was no history of injury, and the growths had been present for 10 years and 3 months respectively.

As to the cause of the growths in Cases I and II we are uncertain. It is possible, though we do not think it likely, that they are congenital and of the nature of the so-called *dermoids*. But they differ from the latter, in that they have neither hairs nor glands, and only in Case I does the growth show any sign of papillæ.

As already mentioned, there were none of the recognised appearances of xerosis of the conjunctiva. The site of the growth was not that usual in xerosis, the mass had not a frothy appearance, the surface could not be scraped off, there was no night blindness, and the patients were in perfect health.

Hocquard has described two groups of cases in his paper entitled "Plaques épithéliales da la Cornée," published in "Archives d'Ophth.", 1881.

(i) Emulsion patches, which apparently are similar or closely allied to xerosis.

(ii) Hypertrophic epithelial patches, occurring on the corneæ of injured eyes subjected to continued irritation.

From his description the latter would correspond with the white thickened epithelial patches seen fairly commonly on staphylomatous corneæ.

We think that our cases are closely allied in microscopic structure to Hocquard's second group, but these were *corneal*, whereas ours are *conjunctival*.

Jessop, in the XVI vol. of "The Transactions of the Ophth. Soc.," describes a case of a small growth on the ocular conjunctiva in a boy, similar to our Cases I and II, except that his growth was red and ours were yellow, but this difference might be accounted for by greater vascularity in the underlying tissue. The late Professor Kanthack examined microscopic sections of it, and described it as consisting of extreme thickening of the surface epithelium of the conjunctiva and increase of the papillæ.

The chief interest of Cases III and IV is, we think, their clinical likeness to epithelioma of the conjunctiva. Case III was thought by one of us to be very possibly epithelioma, and Case IV was seen by several surgeons at the Royal London Ophthalmic Hospital, and also thought possibly to be of a similar nature.

The correct diagnosis was made clear by the microscopic sections; but in the light of subsequent experience, we think that both cases could probably have been correctly diagnosed clinically by the two following points:—

(1) That they had not got the convoluted raised border so characteristic of epithelioma of the cornea and conjunctiva, and (2) apart from the few pink spots seen in Case III—the growths were of the dead-white colour of sodden epithelium, instead of being pinkish white with distinct vessels in the substance of the growth as in epithelioma.

We find great difficulty in classifying these growths satisfactorily. We cannot call them *warts of the conjunctiva*, because they do not show the essential feature of warts, viz., hypertrophy of the papillæ; nor can they be called *corns of the conjunctiva*, as there is no ingrowing process of the epithelium.

They seem to us to correspond most closely with *callosities*

CASE II.

FIG. 4.

CASE III.

FIG. 5.

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CASE I.

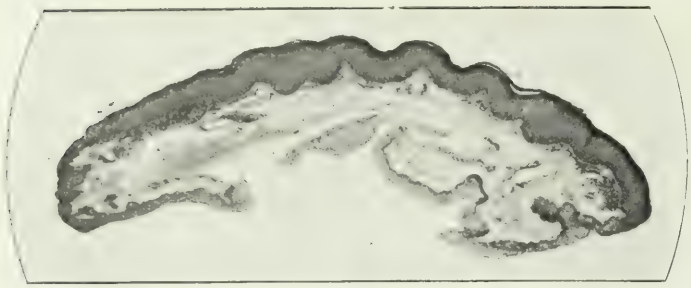

FIG. 1.

CASE III.

FIG. 2.

CASE I.

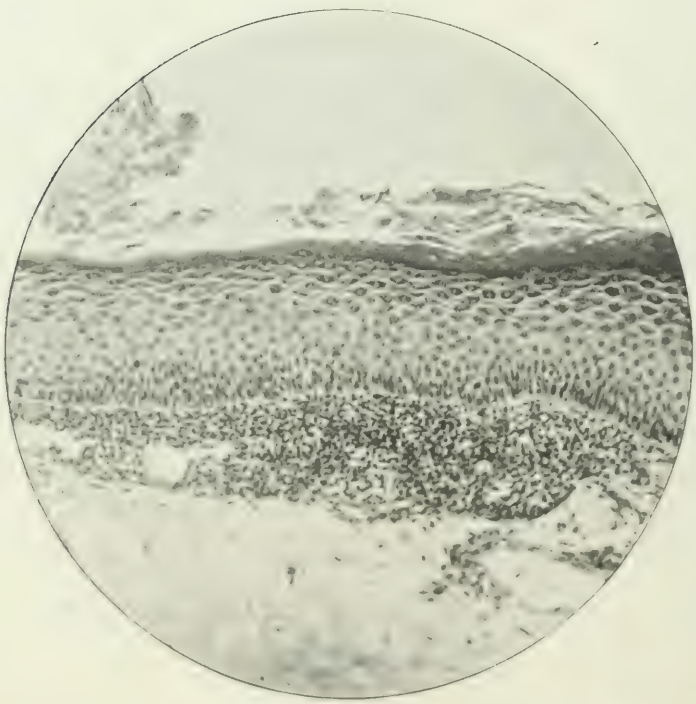

FIG. 3.

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of the skin, which are produced by hyperplasia of the horny layers, and may be either congenital or acquired.

We think that Cases I and II are probably of a different type from III and IV, but till we can gain further information, we class them together under the same title of *epithelial plaques of the conjunctiva.*

In conclusion, we have to thank Mr. Treacher Collins for kindly allowing us to make use of Cases I and II, which came under his care, and Mr. William Lang for allowing us to mention Case IV.

Bibliography.—Beyond the papers already mentioned, we are unable to find any further references to cases similar to those described.

Description of Plates.

Plate 1.

1. Case I.—× 10. Showing papilla and sharp limitation of growth.
2. Case III.—× 15. Showing the great hypertrophy of the epithelial layer; gradual thinning of the edges and the blood vessels embedded in the epithelium. No definite processes of fibrous tissue pass into the epithelial mass and no processes of epithelium pass into the subjacent tissue.
3. Case I.—× 80. Showing the superficial horney layers, stratum granulosum and infiltration of subjacent tissue with small round cells.

Plate 2.

4. Case II.—× 80. Showing the sharply-defined margin of growth.
5. Case III.—× 80. Showing the thin-walled blood vessels embedded in the epithelium and the processes of horny cells passing from the superficial layer into the underlying epithelium.

A PATHOLOGICAL REPORT ON A CASE OF MOOREN'S ULCER OF THE CORNEA.

By W. T. Lister, F.R.C.S.

Through the courtesy of Mr. Lang, I am enabled to bring forward the following case of Mooren's ulcer, which came under his care at the Royal London Ophthalmic Hospital, and to give a pathological report on the condition.

D. H., male, æt. 48, came to the hospital complaining that the R. eye had been painful on and off for 12 months.

There was a mucopurulent discharge on the R. side, and the R. cornea was completely opaque, vascular, and rough. On the inner side of the cornea there was an irregular marginal ulcer with overhanging edges. The vision in this eye was reduced to perception of light only. The L. eye was normal, and vision was 6/6. Owing to the prolonged suffering of the patient, and to the bad state of the R. vision, enucleation of the R. eyeball was performed.

On section of the eye, after fixing and freezing, the anterior chamber was found normal and the pupil was free; the lens and vitreous were clear; and the retina and choroid were *in situ*. Microscopic sections of the cornea were made, which show well the peculiar characteristics of this form of ulceration.

The most striking features of the ulcer itself are its depth and the undermining of the edges. The depth of the ulcer varies in different places from one-third to one-half the thickness of the cornea. The floor of the ulcer presents signs of interstitial inflammation with vascularity, but the amount of infiltration with small round cells varies considerably; in places the floor is fairly clean, in other parts the corneal lamellæ are covered with a layer of broken-down

tissue densely infiltrated with cells. The deepest layers of the cornea show little or no change.

The extent to which the margins are undermined is considerable, varying from $\frac{1}{2}$ to 1 mm. The anterior lamellæ of the cornea, forming the overhanging edges of the ulcer, consist of about one-third the thickness of the normal substantia propria. The epithelial layer has a very irregular base line, and has to a slight degree grown round the free edge. Bowman's membrane is absent, and the corneal lamellæ are greatly altered from the normal. No definite layers can be made out, several blood-vessels are seen cut across, and there is infiltration with round cells, which in places is very dense.

Over the rest of the cornea, where the ulceration has healed, the epithelium is very irregular in thickness, and in places passes in finger-like processes into the corneal tissue beneath. (The plate shows this feature well, and its likeness to rodent ulcer of the skin is easily seen.) At the base of one of the overhanging edges is an oval mass of epithelial cells. This is evidently due to the ingrowth of epithelium at a neighbouring area of ulceration. Bowman's membrane is completely absent, showing, probably, that the whole surface of the cornea has been invaded at one time by the ulcerative process.

The substantia propria is, on the whole, thinner than normal, and, for the most part, shows very marked interstitial change—formation of new vessels and irregular scar tissue. The deepest layers are more affected in this part than opposite the floor of the ulcer.

The episcleral tissue is intensely infiltrated with small round cells, and the vessels are dilated and engorged with blood. Descemet's membrane and the endothelium are unaffected; the angles of the anterior chamber are open; the iris, ciliary body, and lens show no pathological change.

As is well known, corneal epithelium rapidly covers any irregularity in the surface of the cornea, whether it be a clean operation wound or the necrotic margins of an ulcer,

and it is therefore easy to understand that, with a chronic form of ulceration, the epithelial cells may grow round the free margin into the deeper layers of the cornea—and, after cicatrisation has taken place, the embedded epithelium appears on section in the form of finger-like processes invading the underlying tissue.

Some of the sections show that the ulcer is attacking parts of the cornea which have already cicatrised. This may not be usual, since it is at variance with the rough clinical observation, that the ulceration does not, as a rule, return to a part of the cornea which has once healed.

(A complete bibliography is given in Mr. Nettleship's paper in the "Transactions of the Ophthalmological Society," vol. xxii, p. 103.)

Description of Plates.

Plate 1.

1.—Ulcer as a whole under low power × 15. The separation of Descemet's membrane and the splitting of the substantia propria is artificial.

2.—The overhanging edge of the ulcer × 80. The epithelium is very irregular in thickness and extends round the tip. Beneath there is great interstitial change—infiltration with round cells and vascularisation.

The floor of the ulcer is covered with broken-down corneal tissue infiltrated with round cells. On the right side a round mass of epithelial cells is seen.

Plate 2.

3.—The outer edge of the ulcer × 80, showing greater infiltration with round cells.

4.—Healed portions of cornea × 80, showing the finger-like process of epithelium embedded in the cicatrised substantia propria.

Fig. 1.

Fig. 2.

Fig. 3.

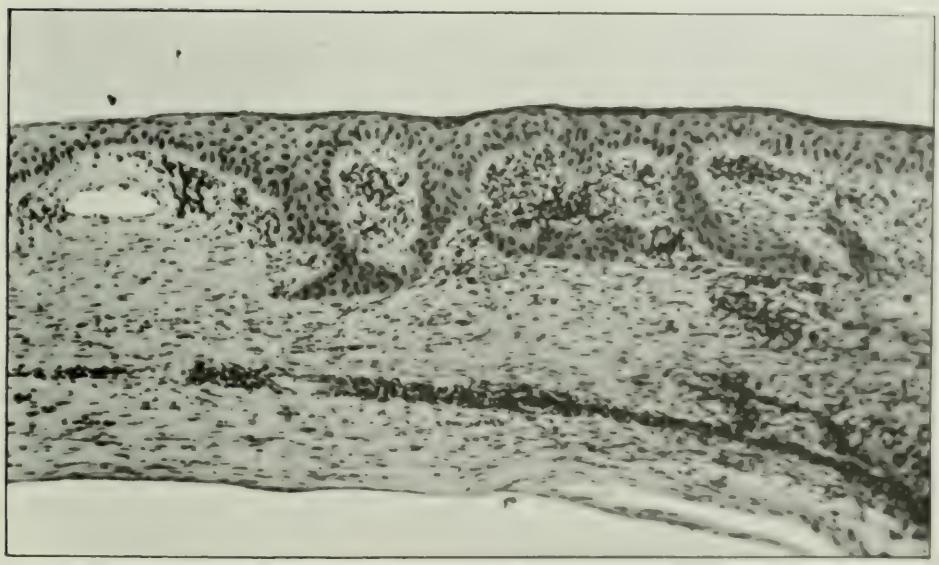

Fig. 4.

TWO CASES OF PIGMENTATION OF THE RETINA.

By E. Erskine Henderson, F.R.C.S.

The following cases show different types of pathological pigmentation of the retina, complicated in each case by glaucoma. In the second case there was retinitis pigmentosa in the other eye.

Case I.

E. R. (6,502), female, æt. 72, was admitted to the R.L.O.H. on 18.xii.02, under Mr. Fleming. She had suffered great pain in the R. eye for one month. She stated that the pain preceded the blindness.

18.xii.02.—The R. eye was in a condition of absolute glaucoma and was enucleated.

L. V = 6/24 H.M. 1·75 d. sph. = 6/6; fundus normal.

R. eye was hardened in 10 per cent. formol, frozen, and bisected vertically.

Macroscopic Examination.—The angle of the anterior chamber was blocked by the iris for a distance of about 1 mm., and there was some irregular pigmentation of the fundus.

Microscopical Examination.—Sections cut in celloidin were stained in hæmatoxylin and eosin by Van Gieson's method. Iron was tested for by Perl's ferrocyanide reaction. Sections were also bleached with permanganate of potash and oxalic acid and subsequently stained.

Cornea.—Epithelium, slightly loosened and œdematous; substantia propria and Descemet's membrane show no changes except at the periphery, where the iris is adherent.

Iris.—Thinned, atrophic, and deeply pigmented. Numerous round cells carrying pigment are on the anterior surface and in the substance. It is adherent to the cornea at the periphery for a distance of about 1 mm. Here some pigment is present in the substance of the cornea and in the meshwork between the angle and the canal of Schlemm.

Ciliary Body.—This also is much atrophied; tubular pigmented processes spread out into the vitreous. [Alt. Treacher Collins.][1]

Lens.—This was not examined.

Choroid.—Atrophic. It is very deeply pigmented in places. Choroid and retina are in many places firmly attached, and here Bruch's membrane shows some thickening. Bruch's membrane is, however, intact. The chorio-capillaris is entirely destroyed. The larger vessels show hyaline thickening of the walls and proliferation of the intinia. Some pigment is to be seen in the inner layers of the sclera, chiefly in the neighbourhood of the short posterior ciliary vessels.

Retina.—Almost all trace of the original structure has disappeared. It is much thinned. The connective tissue framework has undergone extensive proliferation. There is no trace of a rod and cone layer. Between the fibres spaces are to be seen containing a granular material, staining faintly with eosin. Numerous round nuclei are scattered irregularly throughout the entire structure. The pigment epithelium is sometimes depigmented, sometimes wanting, and in other places has proliferated. From these latter places processes are to be seen running into the retina, especially in the neighbourhood of the larger blood vessels. The pigment is mostly intracellular, but numerous fine granules lie free in the tissue. Only in a few places does the pigment pass beyond the hyaloid and form a layer on its inner surface, as in the cases described by v Hippel[2]. In other places there are a few free red blood corpuscles lying on the inner surface of the hyaloid. The walls of the larger blood vessels show much hyaline thickening; in some the lumen is apparently obliterated. These changes affect the whole retina, but are more marked at the posterior pole. All the pigment is bleached by the method described, and no iron reaction is obtained.

Case II.

D. F. (6,522), male, æt. 21, was admitted to the R.L.O.H. on 17.xii.02, under Mr. Lawford.

L. eye had been blind for some years, but had recently been getting larger and painful. It was in a condition of absolute glaucoma. The cornea was opaque, and there was a ring of ciliary staphyloma.

R. V = 6/60 unimproved—corneal nebulæ—nystagmus—shallow A.C. Pigment on lens capsule. The fundus showed the appearance of retinitis pigmentosa. There was a history of nyctalopia of at least 5 years' duration.

17.v.02.—The L. eye was enucleated and prepared for examination in the same way as the first case.

Macroscopic Examination.—The cornea is opaque and thickened There is a ring of ciliary staphyloma. An anterior synechia is present. There are pigmentary changes in the fundus.

Microscopic Examination.—Cornea. This is in a condition of pannus degenerativus. The epithelium is much thickened, and the superficial cells in many places considerably flattened, but all possess nuclei. Vessels of new formation are present in the deeper layers. Bowman's membrane has almost entirely disappeared. The substantia propria contains numerous vessels, and is infiltrated with small round cells. Descemet's membrane is much thickened, and in the neighbourhood of the anterior synechia appears to split into two layers. The endothelium has in some places proliferated and in others has disappeared.

Iris.—Deeply pigmented on the posterior, but deficient in pigment on the anterior surface. Foci of small-celled infiltration are present. A few large hyaline cells, staining deeply with eosin, are present. The pupil is blocked by an endothelial-clad membrane,which extends up to the anterior synechia and becomes continuous with it. The endothelium is probably derived from Descemet's membrane.

Lens.—The capsule at the anterior pole is much thickened and the epithelium has proliferated to form an anterior capsular cataract. The lens fibres underlying are vacuolated and swollen near the equator; the iris is adherent to the lens capsule. The capsule at the posterior pole is much thickened, and here also there is vacuolation and separation of the lens fibres, forming a posterior polar cataract.

Choroid.—Much atrophied; the chorio-capillaris has practically disappeared. The larger vessels show considerable hyaline thickening of their walls and proliferation of the intima. Bruch's membrane is everywhere intact, but shows occasional thickenings.

Retina.—This is partly detached from the choroid as a result

of the hardening process. In some places it is still firmly attached, without any definite fibres passing from one to the other. The neuroglia has largely proliferated, forming a network with spaces containing round cells and some structureless, slightly granular material. Over the greater part of the retina only one nuclear layer is to be recognised. This, on being traced to the macula, evidently corresponds with the inner nuclear layer. Only in the immediate neighbourhood of the macula is a definite rod and cone layer still preserved. In the rest of the retina the outer layers are fused into a fibrous meshwork with arcade-like spaces containing round cells, pigment and finely granular material. The pigment epithelium has in places proliferated, is absent in others, and in yet others has lost its pigment. Processes from the proliferated pigment epithelium pass into the retina and proliferate in the neighbourhood of the larger vessels. The pigment is for the most part intracellular, but also lies free in the form of fine granules in the previously noted fibrous meshwork. The blood vessels are much altered. The walls are thickened and hyaline, the intima has proliferated, and in some the lumen is obliterated and filled with pigment. The optic disc is cupped, but there is not much development of fibrous tissue. As in the preceding case, all the pigment is bleached, and no iron reaction is obtained.

The pathological changes in these two eyes correspond fairly with the classical descriptions of retinitis pigmentosa (Ginsberg).[3]

The first case differs in some respects, notably in the excessive choroidal changes. The fact also of the other eye being free from pathological change, makes the diagnosis of retinitis pigmentosa very improbable. The case was probably one of chorio-retinitis. Stein,[4] in a paper on the pathological anatomy and differential diagnosis of syphilitic chorio-retinitis, describes four cases, two of which were cases of retinitis pigmentosa and two were cases of syphilitic chorio-retinitis. He points out that in both affections we have to do with a somewhat similar change in the choroid and retina, and that this, in his opinion, is due to a wide-

spread sclerosis of the retinal and choroidal vascular system. As points of differentiation, he notes that, in the syphilitic cases, Bruch's membrane is not usually intact, and that the choroid suffers much more than in retinitis pigmentosa. In the latter Bruch's membrane is always intact.

The second case in all probability was one of retinitis pigmentosa.

In both cases there are very extensive changes in the choroidal vessels. They would thus tend to support the theory that attributes the origin of pigmentary changes in the retina to interference with the choroidal circulation. In this connection the experiments of Wagenmann[5] are of importance. In his experiments rabbits were used. He divided various ciliary and retinal vessels, and made careful opthalmoscopic and microscopical examinations of the changes that ensued.

The experiments that chiefly bear on the points at present under discussion were those in which one long ciliary and some of the posterior short ciliary branches on the same side were divided. Half-an-hour after the section an opacity begins to appear in the periphery of the retina of the half whose circulation has been interfered with. In 6 hours this has spread over the whole half retina and remains unchanged for 2 days. As it begins to recede, the retina, owing to folds, shows network-like markings, and fine spots of pigment make their appearance.

From the 5th day onwards larger clumps of pigment are to be seen. The retinal vessels remain normal. After the 4th week some slight atrophy of the nerve is apparent, but is not marked. Later, anterior and posterior polar cataracts are met with. Microscopically, in the early stages there is general serous œdema of the retina, and the outer layers are separated by a finely granular albuminous material; after the 2nd day the rod and cone layer disintegrates. The pigment epithelium in places disappears, and in places proliferates and sends processes into the retina. The retina later becomes very thin and atrophic. No pigmentary

changes follow interference with the retinal vessels. In discussing the bearing of these experiments, the author points out that, owing to the difference between the retinal blood supply in man and the rabbit, too close an analogy must not be drawn (Parsons).[6] In a subsequent paper[7] the same author describes the microscopical anatomy of a case of retinitis pigmentosa, in which the changes in the choroidal vessels support his view. A similar case is reported by Bürstenbinda.[8]

In this case, however, a perforating corneal ulcer had started a panophthalmitis, to which some of the changes observed in the choroid may have been due. Nettleship[9] has pointed out that the somewhat less efficient blood supply to the equator of the eyeball may determine the starting point of the disease. It is difficult, however, to see why the subjects of this disease, if it was entirely due to vascular disturbance, should have only the choroidal vessels affected, and not those in other parts of the body.

Another point of interest in these two cases lies in the possible relation between the retinal and choroidal changes and the glaucoma. E. v. Hippel (*loc. sup. cit.*) reports two cases of glaucoma in which pigmentary changes were found in the choroid and retina. In these, although the angle was free, the fibrous mesh-work between it and Schlemm's canal was filled with pigment cells. He suggests that the pigment cells may stop the filtration process, and that, as happened in his cases, neither myotics nor operative measures will effect a reduction of tension. He quotes similar cases recorded by Priestley-Smith,[10] Alt[11] and Dolganoff.[12] He also points out that pigmentary changes in the retina, similar to those in retinitis pigmentosa, may be present without change in the choroidal vessels as a result of a chemical influence, *e.g.*, a splinter of iron. In neither of the two cases here reported was there much pigment in this situation.

Weiss[13] instances a case of retinitis pigmentosa complicated by glaucoma, and collects from the literature of the

subject 17 others. He points out that it is possible that the cases of glaucoma complicating retinitis pigmentosa are more common than is supposed. Patients with an incurable disease like retinitis pigmentosa drift out of observation, and even if later attacked by glaucoma, attribute this to the original complaint, and as they are already blind do not again appear for inspection.

In the Clinic at Giessen, out of 55 cases of retinitis pigmentosa observed since 1890, only one was complicated with glaucoma.

Birnbacker and Czermak[14] have suggested that an inflammatory process in the choroid may lead to blocking of the venæ vorticosæ and so to glaucoma.

That a similar predisposition to vascular changes may underlie both complaints is suggested by a paper by Blessig.[15] An account is here given of a family of nine, in which two sisters suffered from glaucoma and two brothers and one sister from retinitis pigmentosa.

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I. A CASE OF ORBITAL WOUND IN WHICH THE OPTIC NERVE AND CENTRAL RETINAL VESSELS WERE DIVIDED.

By J. Herbert Parsons, *Curator*.

Records of cases of division of the optic nerve anterior to the entrance of central retinal vessels are rare. Such cases present ophthalmoscopic appearances which are characteristic, and are of diagnostic importance.

Henry Aston, æt. 28, was stabbed in the R. eye with a table-knife on 1.ii.1903. He was treated by Dr. Herbert Hall at the Watford Hospital on the same night. The eye was protuberant, of stony hardness, and quite blind. No temperature developed, the swelling went down, and the tension steadily diminished.

9.ii.1903. Patient admitted to R.L.O.H., under Mr. Lang. There was a wound in the lower lid just below the inner canthus. The eye movements were good in all directions except outwards, the external rectus being paralysed. Slight proptosis. There was extensive subconjunctival hæmorrhage on the outer side. The cornea was anæsthetic; a.c. normal. The pupil was dilated, did not react directly to light, but gave very slight movements in the lower and outer quadrant on consensual stimulation. On ophthalmoscopic examination a large area of œdema was found at the posterior pole; it extended farthest on the outer side, where it reached two-thirds of the distance towards the periphery. The optic disc was pink, with a well-defined physiological cup; the edge of the disc was blurred. The vessels were of good size, the veins slightly larger than the arteries, and both were of nearly the same colour, which resembled that of normal veins. They could be traced to the bottom of the cup, with little variation in size. There was no spontaneous pulsation in either arteries or veins. On moderate pressure by means of the finger upon the globe, the blood could be driven out of both sets of vessels towards the disc. The blood in them was therefore under much reduced intravascular pressure, and the circulation must have been

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extremely sluggish if present at all. A dense opaque white area extended outwards and a little downwards from the outer edge of the disc to beyond the position of the macula, measuring three discs' diameter horizontally, and half a disc's diameter vertically. The edges were sharply defined, and greenish-white; they were less well defined at the inner and outer ends, shading off here into the surrounding œdematous retina. The surface of the patch could be seen with + 2 D, the rest of the fundus being emmetropic, but measurement could not be made absolute as there were no vessels, &c., upon the surface. There was a small, irregular, stippled red spot above the inner end of the white patch, at about a diameter and a half from the disc. The small vessels near the patch were irregular in calibre; they disappeared at the margin of the patch, except at the outer part, where they could be traced as fine lines, often broken, over the surface. Vision, nil; tension normal.

12.ii.03. Appearances much the same. The vessels empty rather less easily on pressure.

13.ii.03. Consensual pupil reaction down and out confirmed by several observers. Slight pressure on the globe produces arterial pulsation, rather more empties the vessels. The similarity in colour of the arteries and veins is slightly less marked. The red spot above the white patch is undoubtedly a punctate hæmorrhage.

16.ii.03. There are many small punctate hæmorrhages, especially beyond the outer end of the white patch. The œdema is clearing up considerably, and does not extend far beyond the patch; the periphery of the fundus looks quite normal. There are vessels on the white patch, with short broken columns of stagnant blood. There is also an appearance of fresh vessels here.

19.ii.03. The pupil contracts with $\frac{1}{4}$ per cent. eserin.

20.ii.03. The œdema has almost disappeared. The white patch is covered with small broken vessels, containing very dark blood. A thin white film passes over one of the lower macular arteries. The hæmorrhages have disappeared. Patient discharged.

5.iii.03. Cornea anæsthetic; small ulcer on outer side, which stains with fluorescein; ciliary injection. Pupil semi-dilated. Slight tendency to ectropion of lower lid opposite site of wound.

16.iii.03. Ulcer *in statu quo*. Ophthalmoscopic examination difficult. The retinal vessels were seen to be irregularly full; some veins were larger, others smaller, than the corresponding arteries; none were fully distended. Fundus bluish, with stippled appearance over the area outside the disc. There is a network of small vessels over the area which was formerly white. No hæmorrhages.

7.v.03. Movement of the eye outwards is now almost full. Ulcer still stains; cornea anæsthetic. Pupil dilates more above than below. Optic disc very pale, obviously atrophic. Retina slightly hazy. Macular region now red, with a spot of pigment at the macula.

18.v.03. Ulcer healed.

4.vi.03. Cornea still anæsthetic. No p.l.

20.vii.03. Eye movements good in all directions, not quite full outwards. Cornea anæsthetic, nebula. Iris immobile; pupil 4 mm. in diameter. Optic disc chalky-white. Arteries and veins equal and small, rather dark, with very little difference in colour, but quite distinguishable. Retina good colour, but much degenerated, showing increase of fibrous tissue. There is a small black ring at the macula. No p.l.; tension normal.

Wounds of the optic nerve have been discussed, *inter alia*, in the monographs of Yvert (1880) and Praun (1899). I propose to consider only those in which the nerve was divided anterior to the entrance of the central retinal vessels.

v. Graefe (1859) reported the case of a student who received a wound from a duelling sword (Schläger). The eye was blind, and ophthalmoscopic appearances resembled those of embolism of the central artery. v. Graefe diagnosed tearing of the central vessels.

H. Pagenstecher (1869) gave a very detailed description of the case of a girl, æt. 12, who was wounded by the sharp iron point of a bar (Stange). Seen on the 4th day after the injury, there was complete amaurosis. Instead of the usual red reflex, there was a bright white reflex from the widely-dilated pupil. The disc was only recognisable by a blood

vessel which ran downwards for a distance less than a papilla-diameter. The vessel contained blood at intervals, was irregular in diameter, and it was uncertain whether it was an artery or a vein. A similar smaller vessel ran upwards. The papilla was bright white, the area between it and the macula grey, fading off towards the periphery to red. Observations were continued for 2 months. On the 5th day the œdematous area was larger; a few more vessels were visible. On the 6th day the œdema had diminished, and the edges of the disc were just distinguishable. More vessels were to be seen; they still showed empty spaces. On the 7th day the œdema was still less; the edges of the disc were distinct, but the area between the disc and macula was still sharply limited from the surrounding retina. On the 8th day the vessels were still fuller; some "choroidal" plaques at the periphery showed commencing pigmentation. This pigmentation continued to increase, more and more pigmented areas being developed, so that they encroached on the central areas. The disc became completely atrophic. The blood vessels continued to increase in number and became better filled. It was impossible to distinguish between arteries and veins. Pressure on the globe caused no pulsation. Many of the vessels were obviously of new formation, and not merely the normal ones restored; this was further proved by the development of anastomotic branches. A month after the injury there was no consensual pupil reaction; the pupil contracted under eserin. The vessels were now smaller than before, and pigment was distributed along them in the peripheral parts of their course. There was at this period a distinct extravasation of blood a little above the macula. At a later stage the peripheral ends of the vessels became reduced to bloodless white cords running through bands of pigment. In 6 weeks the white atrophic papilla was partially pigmented and free from blood-vessels. The retinal blood-vessels appeared a short distance outside the edge of the disc; they were much reduced in size.

H. Pagenstecher in the same paper recorded the ophthal-

moscopic appearances in a case of extirpation of an orbital growth, the eye being retained, though the optic nerve was cut through. On the 4th day there was a white reflex, with a few red vessels; the papilla was indistinguishable from the retina. On the 13th day the neighbourhood of the macula seemed to be detached. On the 18th day the disc was reddish, the vessels distinct, but arteries and veins could not be differentiated. The retina around the disc was still greyish-white, and there were deposits of pigment about the macula.

The observations in this case are amplified in a similar one recorded by Knapp (1874). An extradural endothelioma of the optic nerve* (described by Knapp as a carcinoma) was removed from a woman, æt. 40, the globe being retained. On the 2nd day there was a whitish-yellow reflex. On the 3rd day the tension was normal. On the 4th day the central part of the fundus was white, the periphery yellowish-red. On the 5th day two dark red streaks marked the position of retinal vessels: they were apparently veins; they had pointed extremities and no pulsation was visible. On the 6th day the œdematous area showed diminution; the two vessels were longer, and four or five new small ones had appeared. On the 7th day day there were 15–20 vessels, only two of which reached the centre; these were broadest and longest and were very wavy. The general appearance resembled embolism of the central artery of 5 or 6 days' duration. On the 8th day the contours of the disc were dimly visible, and arteries and veins could be differentiated. On the 9th day venous hyperæmia was more pronounced; there was no pulsation on pressure; there was no dark red spot at the macula. From the 10th to the 12th day the œdema diminished and the venous hyperæmia increased. On the 15th day the latter was very striking, the veins being varicose at the disc. This condition increased still further, with the development of extensive extravasations of blood. Still later the hyperæmia gradually diminished, the disc became pale, and the vessels

* See Parsons, Trans. Ophth. Soc., vol. xxiii, 1903.

were reduced to white strands. The hæmorrhages were replaced by pigment. The author gives three most instructive ophthalmoscopic plates, showing the appearances before operation (optic neuritis), and 3 weeks, and 7 months after operation.

Just (1873) reported a case briefly; red vessels were seen, on a bright white ground, on the 3rd day after the injury. It is improbable that the vessels were divided in this case.

Hirschberg (1879) saw a patient whose optic nerve and central vessels had been divided by a splinter of wood 4 weeks before. There was a bluish mass in the vitreous, probably following hæmorrhage. The retina, where visible, showed hæmorrhages and streaks of pigment.

Aschmann (1884) recorded two cases. In the first, due to injury with a bean-prop (Bohnenstickel), the optic nerve was torn from the retina. The site of the disc looked grey, and formed a deep cup. In the second case, due to a blow with an umbrella, the disc was covered with blood clot, the retina was detached, and the choroid ruptured. After enucleation it was found that the posterior part of the globe was torn.

Karafiáth's case (1884), due to a revolver shot, 22 days after the injury showed blood in the outer half of the vitreous and cupped steel grey papilla, without vessels. Around the disc was a yellowish-white area, reaching almost to the equator on the temporal side, sprinkled with bright and dark brown spots. Beyond this were many hæmorrhages; some dark vessels could be seen only on the nasal side.

Schliephake (1888) reported a case of injury with a billiard cue. The fundus was grey, the papilla hazy, without vessels. The vessels filled up and became congested, but were again scarcely visible after 6 weeks, when the fundus was pigmented.

Liebrecht (1891) recorded a case of partial section of the optic nerve, including the central retinal vessels, by

a piece of glass. The whiteness of the fundus is noted, and the narrowness of the arteries after a few weeks. There was also pigmentation. The early stages could not be observed.

Blessig (1898) reported two cases: (1), Seen on the 2nd day, showed œdema of the retina, arteries narrow, veins beaded, red spot at macula, and after 10 days, disc atrophic, arteries narrow. (This is a doubtful case.) (2), Seen after 4 weeks, showed vitreous opacity, disc pale, vessels narrow, œdema of macula, with red spot.

Mendel's case (1899) gave the picture of embolism of the central artery, and ended in optic atrophy.

Neuburger (1901) recorded a case due to fall on a stick. Seen on the 3rd day, there was œdema of the disc and retina; no arteries were visible, and the veins were irregular. After 3½ weeks the nerve was atrophic, the retinal vessels were partially restored, the œdema had disappeared, and there was a rupture of the choroid.

Birch-Hirschfeld (1902) has reported the most recent case. A boy, æt. 13, was wounded with a hay fork. The case was observed from the 4th day to the end of 3 months after the injury. On the 4th day the tension was diminished; the retina was hazy, with hæmorrhages, chiefly near the nasal side of the disc; the disc was replaced by a hole, due to the nerve having been torn out of the sclerotic; a small vessel disappeared over the edge down and out; other rudimentary vessels were only to be seen at the periphery (Dr. Zetzsche). On the 13th day the cornea was sensitive; pupil was widely dilated, and there was no consensual reaction. The situation of the disc showed a deep cup (−6 D), and was bluish-grey with an evident shadow. The edge of the disc was slightly raised, and showed whitish spots and stripes. The retinal vessels looked fairly normal, of medium size. The area between the disc and to two papilla diameters beyond the macula was grey, fading off into the normal retina outwards and above, but limited below by a narrow area of hæmorrhage. In the next 4 days

the œdema at the posterior pole cleared considerably, but was still visible on the 21st day. In the course of the following 2 months, the cup at the position of the disc became filled with white fibrous tissue, and bands of the same material replaced the hæmorrhagic area, with the exception of a dark red spindle-shaped patch. The fibrous tissue at the disc sent bands forward into the vitreous. There was a little pigmentation below, and less above, the disc.

Division of the optico-ciliary bundle experimentally was first investigated by Berlin. He operated upon rabbits, and found very extensive œdema of the whole retina. Leber confirmed the results in the cat. It is not easy to divide the central vessels in lower mammals, as they enter the eye close to the sclerotic.

The experiments were extended by Krenchel, who operated on frogs, and the whole subject was very exhaustively investigated by Wagenmann. The latter found that simple section of the nerve and central vessels did not cause retinal œdema, but this followed section of the ciliary arteries only. This observation requires confirmation. Wagenmann also attributes the pigmentation of the retina to injury of the ciliary blood supply.

A review of the cases presents a very constant group of ophthalmoscopic signs, which are of extreme diagnostic and pathological interest. Many of the signs, such as disappearance of the vessels, temporary diminution of intraocular tension, &c., are only what might be expected. Two features demand special consideration: (1) Œdema at the posterior pole; (2) re-vascularisation of the retina.

Œdema at the Posterior Pole.—The occurrence of œdema in tissues deprived of their blood-supply is exemplified in many conditions in various parts of the body, and need, therefore, occasion no surprise in the retina in such conditions as embolism of the central artery or section of the optic nerve anterior to the entry of the central retinal vessels. Anæmic tissues take up fluid with great avidity,

and this is doubtless due to the alteration in osmotic pressures brought about by the retention of katabolic products, though the exact rationale of the phenomenon has yet to be worked out.

It is less easy to understand why the macular region should suffer in so disproportionate a degree. The resemblance of the ophthalmoscopic picture in these wounds of the nerve to that of embolism has been pointed out by several observers. There can be little doubt that the cause is the same in both conditions. Direct injury of the posterior pole of the eye can be definitely eliminated in several of the cases, as in that recorded here. In the wounds of the nerve the whole retina would probably be found to be œdematous in the very earliest stage a few hours after the injury; if not, it must be due to the comparatively free vascularisation of the anterior part of the choroid by the recurrent branches of the anterior ciliary vessels. The exact parallelism between the œdema of the macular region in embolism and that in the condition under consideration is found in the fact that in the latter the macular region is the last to clear up as the retina becomes revascularised. This depends, I believe, upon the fact that, *quâ* retinal blood supply, this area is worst provided for. Hence, as the circulation is re-established, effete material will stagnate there longest, and we have seen that this is the most probable cause of the œdema.

It has already been noted by H. Pagenstecher, in his second case, that the neighbourhood of the macula seemed to be detached. I think it is probable that there was an actual slight detachment of the retina in this situation in the case recorded above. In the earliest condition the bright white summit of the area afforded no means of accurately measuring its position relative to that of the surrounding retina, which, it must be remembered, was also swollen. After the development of new vessels, the difference in height was too slight to authorise a dogmatic statement. Such detachment, if present, was due to the accu-

mulation of fluid under the retina at the most œdematous part. This may account for the absence of a cherry-red spot, but there are other reasons for this. It will be noted, in the cases previously reported, that such a spot is seldom present, though it may be simulated by a hæmorrhage, as in my case. It is mentioned in Blessig's first case, but the condition here of the vessels on the 2nd day is scarcely consistent with division of the central trunks behind the globe.

Reported cases of embolism seem now to have proved conclusively that the red spot at the macula may be due to hæmorrhage, though in most cases hæmorrhage is absent. It would be interesting to discuss the cause of the hæmorrhage when it occurs, but this is beyond the scope of the present paper. When it is absent we must fall back upon the theory of the contrast of the choroid, seen through the thin retina at this spot, with the surrounding œdematous, non-vascular retina. And this is a good reason why there should be no red spot when the optico-ciliary bundle* is divided, even if the retina is not actually detached. Under these circumstances the choroid must be almost emptied of blood, the large veins being submitted to the intraocular pressure, and thus emptied. Some blood will still enter the choroid from the recurrent branches of the anterior ciliary vessels, but the choroid at the posterior pole of the eye will certainly suffer most, and we must remember that the partial anterior ciliary blood supply will prevent the intraocular tension from falling to zero.

Revascularisation of the Retina.—No refilling of the retinal blood vessels can occur until the cut ends of the central vessels behind the globe are sealed up. This doubtless happens quickly through retraction and clotting. As soon as organisation is sufficient to withstand the low venous pressure, the vessels will commence to refill through the direct and the indirect cilio-retinal anastomoses near the optic disc.* These anastomoses are rather venous than

* See PARSONS, The Ocular Circulation. London, 1903.

arterial; hence the veins will fill up first. This is what we have seen actually occurs, and it takes place on the 4th or 5th day. The blood is, of course, derived from the recurrent branches of the intact anterior ciliary arteries, and also from any posterior ciliary arteries which may have escaped section. The retinal arteries probably fill up first from the periphery, the blood trickling in in the wrong direction from the already refilled veins.

As soon as a few vessels have been refilled, new capillaries will be actually created in these areas. The conditions are exactly similar to those which obtain in the vascularisation of an organising clot, &c., with the exception that the inert mass in the present instance already contains pre-formed blood channels. In both cases there is the requisite stimulus for the proliferation of the capillary walls, new endothelial tubes are formed, and these later put on adventitial sheaths, and so anastomatic branches are formed. In this manner, by the continual opening up of the old paths and formation of new ones, the circulation is re-established. The reverse current in the retinal vessels, if it occurs, is probably of short duration, the arterial anastomoses developing rapidly under their greater internal pressure, so that a normal current is set up.

How does the blood escape from the eye? The answer to this question also explains the occurrence of venous hyperæmia and hæmorrhages. The retinal blood can no longer return by the central vein, which is now quite sealed up. It has to pass out by the same anastomoses round the disc. Here it enters the choroidal veins, is carried forward to the venæ vorticosæ, and so escapes. It is obvious that this circuitous course must offer great resistance to the exit of the blood. Further, the more blood that enters the retinal vessels, which are now, moreover, increased in number beyond the normal, the more difficult will it be for the blood to escape. It is naturally forced on by the high arterial pressure into the capillaries and veins; these become

more and more congested, until finally the weaker ones give way, and extravasations of blood take place.

This we have seen to be the actual course of events in some of the cases, but not in all. The reason is not far to seek. The retina has ceased to be a functioning organ; it is now little more than an organising scar. The changes which occur in the new-formed blood vessels, leading to the formation of a connective tissue sheath, do not stop at that point, but go on to the generation of fibrous tissue which replaces the old retinal structures, and eventually, by its contraction, throttles the vessels which produced it. Whilst the process of revascularisation seems to proceed at a very constant rate in all the cases recorded, the process of organisation, which is to be regarded as antagonistic, seems to vary greatly, both in rapidity and in amount. These variations must depend upon the very complex conditions of the individual cases. Hence, in one case we get venous hyperæmia and hæmorrhages; in the other these may be reduced to a minimum, though they are apparently never absent. In the latter event, a moderate hyperæmia passes gradually and almost unnoticeably into anæmia, with narrow vessels which are soon obliterated. The ultimate effect is the same; the retina is transformed into fibrous tissue, in which pigmented areas mark the sites of former hæmorrhages.

Finally, a few remarks may be made about the special case under consideration. What happened is probably as follows:—The knife entered at the lower and inner angle of the orbit, and passed between the internal and inferior recti, pushing these aside without seriously injuring them. It is astonishing how structures may escape a large weapon in this manner, and it would be hardly conceivable if it were not confirmed by innumerable similar cases. The knife then passed on, cutting or tearing through the whole optico-ciliary bundle, with the exception of a few short posterior ciliary vessels and nerves on the lower and outer side. That these escaped is proved by the persistence of the con-

sensual pupil reaction in the lower and outer part of the iris. The section of the nerve is proved by the blindness, absence of direct pupil reaction, and subsequent optic atrophy. The knife, being directed up and out, then caught the VIth nerve on the inner and upper part of the external rectus, near the back of the orbit, and partially or completely divided it. It is unlikely that the muscle was divided, both on account of the nature of the injury and the return of function. For the latter reason it is probable that the nerve may have been merely bruised.

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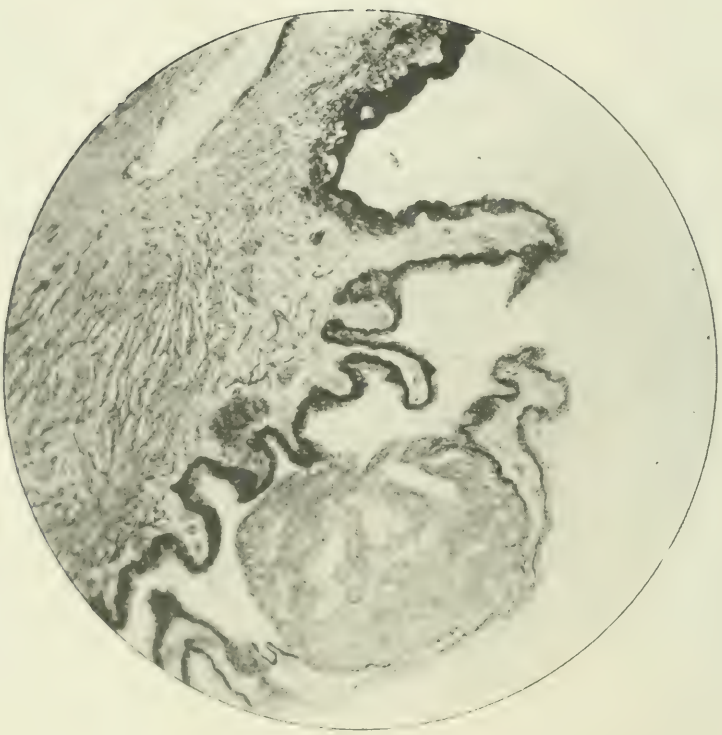

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II. EPITHELIAL HYPERPLASIA OF A CILIARY PROCESS.

THE following case is of pathological interest from the point of view of epithelial growths of the ciliary body. It is almost identical with an "epithelial tumour of the ciliary process" described by Hanke (*v. infra*).

A man, æt. 65, was admitted to the R.L.O.H., under Mr. Holmes Spicer, the left eye having become inflamed 12 days previously. There was no history of injury. The conjunctiva showed intense chemosis; there was a large area of ulceration of the cornea, and this steadily advanced whilst under observation. R. V = 6/36.

It is unnecessary to enter into details of the clinical condition, since the swelling on the ciliary process was probably quite independent of it.

Pathological Examination: Macroscopic.—Large ulcer of cornea with infiltration around. A.c., full of yellow coagulum.

On section in a sagittal direction, an anterior synechia of the iris was noted. The lens was normal, and the retina and choroid were *in situ*.

Microscopic.—Cornea, nearly the whole surface ulcerated; upper and central part entirely necrosed; surface of lower part densely infiltrated in anterior layers. Bowman's membrane is seen for a short distance below; Descemet's membrane extends rather farther below, and is seen for a short distance above. A.c., full of fibrinous coagulum, containing leucocytes; both angles full of leucocytes. Iris, acute purulent iritis; pupil is adherent to slough. Retina and choroid, *in situ;* show congestion and some infiltration.

On one ciliary process below is a globular mass on the anterior surface, with its long axis in the direction of the process. It is about 1 mm. long by 0·75 mm. wide. The non-pigmented retinal epithelium can be traced on to its surface, whilst the pigmented epithelium is continued under it, though the cells soon lose their pigment, which is also absent or scanty on many of the processes.

The main mass of the swelling consists of irregular groups of cells resembling the non-pigmented epithelium, lying in a hyaline matrix, with numerous irregular spaces. The nuclei stain faintly, are round or oval; the outlines of the cells are indistinct. No tubular arrangement of the cells can be demonstrated, though the general disposition is suggestive of such an arrangement.

It is clear, I think, that these cells are due to the proliferation of the non-pigmented or inner layer of the retinal epithelium, and that the proliferation has taken place into the vitreous body, the underlying ciliary process showing the same condition as the others. There is no evidence of inflammation, such as infiltration with leucocytes, either in or around the mass.

The only case on record which at all nearly resembles this one is that of Hanke from Fuchs's clinic. The patient in this case was a woman, æt. 65, and the tumour was similarly met with by accident, the eye being examined for the sake of a pterygium. The swelling was reniform and was covered by both layers of epithelium, though this seems doubtful, both from the figures and the description. In any case, the growth was developed from the non-pigmented layer, and consisted of bands and nests of epithelial cells forming a coarse network. As in my case, the contours of the cells were obscure, the protoplasm having run together into a homogeneous mass, in which the nuclei were embedded.

Hanke discusses the nature of the growth. He regards it as most allied to the epitheliomata. Against this view he mentions the absence of a connective-tissue stroma, and the development of communicating spaces. The first difference is explained by the growth outwards into the vitreous; the second by the specific function of the epithelium of the ciliary processes as a secreting organ.

The absence of tubules, the bands of cells having no lumina, is held to negative the diagnosis of an adenoma of Treacher Collins's glands of the ciliary body, as well as the position of the growth in a ciliary process and its origin from the inner non-pigmented layer.

I think it most probable that both growths are due to chronic degenerative hyperplasia of the epithelium in old age. In Hanke's case the eye was practically healthy, and in mine the disease which led to removal must be looked upon as entirely independent. There are many reasons against regarding the growth as an epithelioma in the ordinary sense of the term. There is no evidence of malignancy, and proliferation is entirely localised, and in a direction outwards. There is no tendency to invasion of other tissues. It might much more reasonably be regarded as a papilloma, and compared with the warty growths which are so common in old age.

Proliferation of epi- and endo-thelium is found in many parts of the eye in old age, and often leads to the formation of warty excrescences. Examples are found in the warty excrescences on Descemet's membrane, more particularly at the periphery. These are undoubtedly due to proliferation of the endothelium and deposition of a hyaline material similar to the membrane itself. More nearly allied to the tumours under consideration are the warty excrescences found on the pars plana in old age. These have been carefully examined and described by Kuhnt and Kerschbaumer. They are due to proliferation of the non-pigmented cells into the vitreous. Neighbouring nodules may cause a festoon-like arrangement. When they are large the central cells may degenerate, and give rise to cysts, which sometimes reach a considerable size.

Similar growths, also in old people, may have a much more pronounced adenomatous type. Such have been described by Pergens and Alt.

The process therefore which gives rise to these unusual growths is essentially degenerative in character, associated with old age. The degenerative changes probably lead to the formation of irritative substances in minute amount, and these stimulate the cells to proliferation. The process is probably extremely slow.

No sharp line can be drawn between these chronic hyper-

plastic changes and the more rapid—but still chronic—ones which lead to the formation of pigmented tubules in cyclitic membranes. Here the irritation is more pronounced, and an essential difference arises in that the epithelium is pigmented, at any rate, in the later stages. (See Treacher Collins.)

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ACUTE DACRYO-ADENITIS.

By William Inman, Senior House Surgeon, Royal London Ophthalmic Hospital.

Affections of the lacrimal gland have received very little attention from English writers, and literature upon the disease is meagre. The leading text books dismiss the subject briefly. Theobald, in Norris and Oliver's System of the Diseases of the Eye, gives the most complete account of dacryo-adenitis. He divides the condition into two forms: chronic and acute, the latter being very rare. He quotes the following authorities:—

1. Arlt, in his "Lehrbuch," 1853, says he has never seen a case of dacryo-adenitis.

2. Power asserts in the Lancet, 1886, that the indices of the Royal London Ophthalmic Hospital Reports make mention of only one case of abscess of the lacrimal gland.

3. Hirschberg states that among 22,500 recorded cases of diseases of the eye, he met with but one case of suppurative inflammation of the gland.

Power's article, referred to above by Theobald, is a report of a series of lectures on Diseases of the Lacrimal Apparatus, delivered at the Royal College of Surgeons. His observations are to the effect that malaise precedes the signs of local inflammation by one or two days. Then the pain begins in the side of the head, radiating towards the parotid, which is usually swollen and tender. After mentioning the condition of the lid and conjunctiva, he goes on to say that resolution rarely takes place, and that the abscess when formed, bursts into the conjunctival sac, or near the outer angle of the orbit. The disease may occur at any age.

A non-suppurative form of acute dacryo-adenitis, termed "mumps of the lacrimal gland," is, according to Power,

rare, and in the single case there quoted, which was recorded by Hirschberg, it was bilateral.

Theobald goes on to say that the disease is probably less rare that these writers indicate, as the diagnosis is not always easy, being often confused with simple orbital cellulitis. Referring to the acute form, he describes the attack as being usually severe, delirium and cerebral excitement having been noted in some instances. The inflammation very often subsides without the formation of pus. When pus does form, it may discharge through the skin, or into the superior conjunctival *cul-de-sac*. The affection is usually unilateral, but not infrequently both glands are simultaneously affected. The globe may be greatly displaced and its movements restricted and painful. Nettleship (St. Thomas's Hospital Reports, 1877) has met with cases of dacryo-adenitis in which the inflammation was confined to the accessory portion of the gland, the main part not being involved.

Galezowski and C. E. Rider have called attention to the occurrence of the disease in connection with mumps.

Fuchs, in his Text Book of Ophthalmology, says: "It is extremely rare for the lacrimal gland to be the seat of disease. Dacryo-adenitis may go on to resolution; in other cases, suppuration of the gland with discharge of the pus externally has been observed, a fistula remaining afterwards. There have even been described cases of bilateral dacryo-adenitis which ran either an acute or more frequently a chronic course; some of these cases were complicated with simultaneous swelling of the parotid glands."

Swanzy mentions that he has seen one case of acute purulent dacryo-adenitis, but no instance of the chronic affection. When suppuration has taken place the abscess may open into the conjunctiva, as it did in his patient, or through the skin.

Lawson, in his recently published work, considers the chronic form to be more common than the acute. One case of the latter which came under his notice had inflammation

of both lacrimal glands simultaneously, the course of the disease resembling an attack of mumps. He says that cases of acute dacryo-adenitis may suppurate, but gives no indication of the frequency of this complication.

An examination of the Hospital records of the past 10 years shows that not a single case of inflammation of the lacrimal gland was admitted as in-patient during that period until 1901, when one case came under observation. In 1902 several patients suffering from this disease were treated. The number of cases treated as out-patients cannot unfortunately be ascertained, but it is probable that the condition is by no means as rare as described. Appended are notes of 10 cases observed during the past 2 years. They are all classed as acute dacryo-adenitis, since in the majority the inflammation was sufficiently acute to lead to abscess formation, and in those cases where resolution took place, the symptoms are marked by some severity and could not properly be described as chronic. Indeed, no case of chronic dacryo-adenitis has come under the writer's notice.

Gertrude F., æt. 15, became I.P. under Mr. Silcock on May 28, 1901. This case was the first to come under the writer's observation. The notes taken at the time were few, and are only of value in showing that suppuration occurred. It was the discovery of this instance which prompted further investigation of the affection.

The patient attended hospital complaining that the L. eye had become swollen and painful 10 days previously. There was much swelling and induration over the region of the L. lacrimal gland. It was described then as having "a soft elastic feel," and fluctuation was obtained.

Under ether, the abscess was evacuated on the day of admission. Ten days later the patient suffered from an attack of follicular tonsillitis. She was discharged on June 15, when there was still slight discharge from the abscess cavity.

Chas. W., æt. 17, admitted under Mr. Collins, August 26, 1902. Began 14 days ago, as small swelling immediately above the outer canthus. Eyes always normal until that time.

26.viii.02. *Condition on Admission.*—Marked œdema of R. lid, with swelling and induration over the upper and outer orbital margin. Injection and œdema of outer part of ocular conjunctiva. Tenderness on pressure. No proptosis or displacement of globe. Movements of eye not interfered with. Upper lid cannot be raised voluntarily. Fluctuation cannot be made out from external palpation. Temperature 99(2)°. Some malaise. General anæsthetic given. Lid can now be everted with some difficulty, and enlarged gland exposed. Its surface is bluish-purple, and there is much congestion. Fluctuation obtained. Outer canthus slit and gland incised at the most prominent point. Much pus evacuated (about a drachm). Boracic ointment used to protect cornea from the discharge, and irrigations of conjunctival sac with perchloride of mercury, combined with fomentations, ordered.

27.viii.02. Swelling subsiding rapidly. Patient feels much better. Much purulent discharge from gland. Pus oozes from opening when pressure is made through lid. No tenderness. Temperature normal.

31.viii.02. Some thickening above outer canthus persists. Lid now easily and painlessly everted. Gland less swollen. No discharge.

1.ix.02. Thickening of lid disappearing quickly.

Jane C., æt. 3, became O.P. under Mr. Lawson, June 29, 1903. When first seen there were œdema of conjunctiva, suffusion, no discharge, and swelling of the L. upper lid. Impetigo of nostrils was a prominent feature. The child was treated as an O.P. until July 2, 1903, when she was admitted as I.P. under Mr. Collins.

History.—Father says that the child first complained of pain on June 25th. A little discharge from the eye was noticed on the preceding day.

The L. *lower* lid appeared to be slightly swollen, and, on the following day the upper lid became enlarged on outer side, and continued gradually to increase in size. No previous attack, or any other affection of eyes. No recent illness.

2.vii.03. *Condition.*—Whole of upper lid œdematous, fading away on inner side; outer part brawny and indurated. Swelling

limited on outer side by external tarsal ligament. Fluctuation cannot be obtained by external palpation. Palpebral aperture closed owing to thickening of tissues of lid. Some tenderness on pressure. No proptosis. Interval can be felt between the swelling and the orbital margin. No enlargement of pre-auricular gland.

R. lacrimal gland normal. Temperature 97°.

Under chloroform the upper lid can be everted, and the palpebral conjunctiva is seen to be normal.

Lacrimal gland enlarged and fluctuating. Posterior limit cannot be made out.

With finger in upper fornix the lateral boundaries of the gland can be felt, although the surrounding tissues are slightly thickened, as well as œdematous. There is no sign of the abscess pointing at any particular spot.

Gland incised, after having been made to present by pressure externally through lid. About a drachm of thick creamy pus evacuated.

3.vii.03. (18 hours after operation.) Marked improvement in external appearance. Much purulent discharge. Swelling subsiding. No tenderness. Temperature normal.

5.vii.03. Outer portion of upper lid thickened. Very little discharge. There is now no œdema of conjunctiva, and only a little injection at the outer side.

10.vi.03. Gland cannot now be felt through the lid.

Leslie F., æt. 11, admitted under Mr. Tay, August 23, 1903.

History.—The L. eye became inflamed 11 days ago, as if a stye were beginning to form. There was redness at the outer end of the upper lid. No affection of the conjunctiva preceded the attack. The inflammation persisted for a week. The patient was then seen by a doctor, who treated the eye with lotion.

The swelling began to subside yesterday. There was much pain until 3 days ago.

No recent illness. Has never had similar attack before. Had operation for adenoids 6 years ago.

Present Condition.—L. Conjunctiva injected and œdematous, especially on the outer side. Lacrimal gland moderately swollen. Skin of lid over the gland is red and thickened.

Little tenderness. No fluctuation. Gland freely movable, and lid can be everted. There is no evidence of an abscess having opened spontaneously. The vessels on the membrane lining the gland are much distended and tortuous.

Globe freely movable. No proptosis.

There is some chronic marginal blepharitis.

Temperature normal.

24.vii.03. Swelling subsiding, under treatment with fomentations. No tenderness. No discharge from conjunctival sac.

30.vii.03. Discharged. Lacrimal gland is now nearly as inconspicuous as that on R. side.

Jack D., æt. 4½, became O.P. under Mr. Tay, March 16, 1903.

History. Became ill with mumps a week ago. Only L. side affected. The following morning the L. eye became bloodshot, followed 24 hours later by some discharge from that eye, whilst a small lump was noticed on outer part of upper lid. On the 4th day the lid was red, tender, and swollen. This morning an abscess appears to have opened into the conjunctival sac, blood and pus being discharged, causing the pain and tenderness to diminish rapidly.

Present Condition.—There is a localised swelling of outer part of L. upper lid. A central nodule is hard; the surrounding tissues are comparatively soft, and outer margin of induration is limited by the external tarsal ligament. There is very little tenderness. Outer part of ocular conjunctiva is much swollen. No fluctuation. Much purulent discharge oozing from gland can be seen. Pre-auricular gland slightly enlarged and tender. L. parotid gland swollen and slightly tender. The swelling extends backwards behind the ear. Patient can now open mouth widely. Temperature 101°.

17.iii.03. Swelling subsiding, but lacrimal gland is still sufficiently enlarged to prevent examination of orbital wall on outer side with the finger. There is still some tenderness.

23.iii.03. No tenderness. Gland moves freely under the finger. The lid is on a slightly lower level than that of the opposite side, at the outer end.

9.iv.03. Gland not palpable.

4.8.03. Has become O.P. again, suffering from severe

phlyctenular conjunctivitis. There is now no indication of the former inflammatory attack.

Sydney B., æt. 6, became O.P. under Mr. Silcock, April 10, 1902.

History.—L. became inflamed ("like a stye") a week ago. The lid was swollen, and the eye was gradually closed. Two days later the condition was aggravated by blow from brother's knuckle.

Present Condition.—L. Trace of conjunctival discharge. Little swelling and redness of lids (especially the upper one), with an indurated mass at the outer extremity of the upper lid. Much œdema of conjunctiva (ocular) on outer side, a fold being pushed out between the lids. No fluctuation or proptosis can be made out. The swelling is tender, and the lids cannot be separated or everted without much pain. Temperature 99$^{(8)}$°.

The mother was ordered to apply fomentations at home. The child was brought to the hospital on the following morning, and as the swelling was not reduced, admission to the hospital was advised.

A general anæsthetic was given, and the external canthus slit. This was rendered necessary by the marked thickening of the lid, and the naturally small palpebral aperture. Upon everting the lid, the enlarged lacrimal gland could be seen as a nodular purple mass projecting downward nearly as far as the edge of the upper lid, but not distended inwards towards the middle line, or downwards below the external tarsal ligament.

An incision was made into the gland substance, and a small quantity of pus evacuated.

On the following day (April 12th) the temperature was normal, and the swelling had subsided to a considerable extent. Three days later the child was discharged.

Flossie O., æt. 21, became O.P. under Mr. Lang, January 26, 1903.

History.—Patient felt a little pain "at the back of the L. eye" 2 weeks ago. There was then a little conjunctival injection.

On January 22nd the upper lid became swollen, hot and tender, but the conjunctiva does not appear to have been

œdematous or shown greater injection than at present time. There was considerable pain. On January 23rd a profuse purulent discharge from the eye began to flow, and the symptoms abated.

There has been no previous attack of inflammation of this or other glands, and no cause can be assigned for this attack.

Present Condition.—The lid is swollen to moderate extent, but the skin shows signs of having been stretched, being now wrinkled and desquamating. There is some tenderness on pressure. The conjunctiva is injected on the outer side, but there is no œdema of this membrane. Upon eversion of the upper lid the lacrimal gland is seen to be enlarged and congested, and pressure of the lid causes pus to exude from a sinus, opening at the most prominent portion of the palpebral part of the gland.

The lacrimal gland of the R. side is normal in size. The L. globe is normal, but the R. globe shows signs of having been severely inflamed at some distant period—probably from ophthalmia neonatorum.

9.ii.03. There is still a little thickening of the lid, and the gland is slightly enlarged. No discharge now.

William V., æt. 34, became O.P. under Mr. Lang, July 28, 1902.

The swelling and pain began 2 days ago.

Present Condition.—Skin over region of L. lacrimal gland is red and swollen.

There is not much pain, but the parts are tender. The lacrimal gland can be felt through the lid as a nodular body, surrounded by indurated tissue. It is easily moved against the frontal bone.

The ocular conjunctiva is injected and œdematous at the outer side, being thrown into folds at the outer canthus. The upper lid cannot be everted, but by gently pulling it forward away from the globe, the inflamed accessory portion of the gland is exposed to view.

Fluctuation cannot be obtained.

28.vii.03. Protruding gland incised, under cocaine, through the conjunctiva. Bead of pus evacuated.

Fomentations every 4 hours ordered.

30.vii.03. Inflammation has subsided: no pain now.

Swelling less marked. Eye is proptosed—proptosis not noted previously.

31.vii.03. Attended hospital. Condition so much improved that further attendance deemed unnecessary.

Arthur B., æt. 37, became O.P. under Mr. Collins on November 3rd, 1902.

History.—A week ago the lids of the L. eye became rapidly swollen and red. There was great pain.

He consulted his doctor, who treated him with hot lotion. The swelling subsided, and the pain was relieved to some extent. Two days later symptoms again developed, more severely than before, and the eye was quite closed by the swelling of the lids.

A gradual improvement then began, with a slight relapse 2 days ago.

A month ago he had an attack of influenza, and was confined to bed for a week, and to the house until a week before the present inflammation.

There is no history of traumatism or constitutional disease.

3.xi.02. The present condition of the patient probably shows a stage of subsidence. The L. upper lid is red, swollen and œdematous. There is much chemosis and injection of the conjunctiva, with a little catarrhal discharge. The lacrimal gland is enlarged and indurated, and manipulation causes a moderate amount of pain.

The pre-auricular and sub-maxillary glands are not swollen.

The globe is normal. There is no displacement or interference with its movements.

6.xi.02. Attended O.P. again. No note made as to condition.

10.xi.02. Swelling less marked, but there is still thickening and tenderness in the region of the lacrimal gland.

13.xi.02. Improvement to such an extent that patient was discharged.

Nelly W., æt. 15, became O.P., May 18, 1903 (Mr. Lang). Slight swelling appeared 14 days ago in outer extremity of L. upper lid. Patient bathed her eye with "boracic lotion and

witch hazel." There was much pain, which continued for several days, but had disappeared at the end of a week. The swelling rapidly increased during first 2 days. She could not open the lids owing to tenderness and stiffness. The conjunctiva was swollen at the outer side. No malaise.

She first attended hospital because she feared a squint might appear owing to the persistent swelling. No history of diplopia.

There has been no preceding affection of the eyes, with the exception of "a slight cold in L. eye, which was watery, but not red." This occurred a week before the onset of the dacryo-adenitis. The R. eye was not watery on that occasion.

There has been no discharge from the eye.

18.v.03. Condition was as follows. Marked swelling of L. accessory lacrimal gland. Upper lid œdematous. Œdema of outer part of ocular conjunctiva, with little increased vascularity. Gland moves freely against the orbital margin. No pain and very little tenderness. Globe moves freely in all directions. No proptosis. It is doubtful whether the posterior limit of the swelling can be distinguished with finger in upper conjunctival fornix, but the indurated tissue becomes thinner as it is traced backwards. R. gland normal.

25.v.03. Upper lid still œdematous. Gland less swollen. No tenderness. Several engorged conjunctival veins at outer canthus.

Patient says that 5 days ago, a "water-blister" developed beneath ocular conjunctiva on outer side. This disappeared yesterday.

4.vi.03. Gland still enlarged. Lid cannot be fully raised.

18.vi.03. Palpebral portion of gland can be felt through lid, and is visible on eversion of lid. No pain or redness.

30.vii.03. No swelling can be now made out. L. upper lid still a little thicker than R. lid. Has pustular blepharitis in R. Does not complain of dryness of L. eye.

The cases described in the literature dealing with this subject divide themselves naturally into two main groups:—

(1) Those in which the affection of the lacrimal gland is associated with mumps. The main features are (*a*) it is usually bilateral; (*b*) suppuration does not occur.

(2) Those in which there is no connection with mumps: the leading features being (*a*) it is usually unilateral; (*b*) suppuration may or may not occur.

In discussing the first group, the writers are agreed upon the course of the disease, but concerning the second, they are strangely at variance. For instance, regarding its characteristic features, Theobald says, "not infrequently both glands are affected," whilst Fuchs only refers to bilateral affection in connection with mumps. Again, in Theobald's opinion, the inflammation very often subsides without the formation of pus, whilst according to Henry Power the condition rarely resolves.

A comparison of the notes shows that this variety of acute dacryo-adenitis has a characteristic form of onset, and runs a definite course, rendering diagnosis easy at all stages. An attack begins with a sensation of stiffness in the outer part of the upper lid. There is some pain and redness, which increase rapidly within the first two or three days. A little conjunctival redness occasionally precedes the onset of the acute symptoms, but in the majority of the cases recorded there was no definite conjunctivitis prior to the inflamed condition of the lid.

The pain, which in the worst cases is severe, is not deep-seated, but is referred to the lid, and in the early stages the patients commonly consider that a stye is forming. This suggests that the palpebral portion of the gland is first involved, the infection spreading backwards to the main part. This feature could, however, be also explained by the swelling of the inflamed structure taking place in the direction of least resistance, &c., towards the front of the orbit. The lid would thus be stretched and the nerves of the conjunctiva pressed upon. A prominent feature frequently at this stage is marked œdema of the ocular conjunctiva on the outer side. This is out of all proportion to the conjunctival inflammation accompanying it, and may be explained by supposing the ophthalmic veins and lymphatics to be pressed upon.

There may be a little catarrhal discharge from the conjunctiva, but it is never profuse.

The lacrimal gland shows distinct enlargement. The surface is nodular, and the vessels lying upon it are seen to be swollen and tortuous, giving the gland a bluish-purple colour. Pressure upon the lid, which causes considerable pain, gives a feeling of general induration of the whole of the outer half of the lid, fading away gradually on the inner side, but being sharply defined on the outer side, being here limited by the external tarsal ligament. This outer limitation is not always visible to the eye, owing to the superficial œdema spreading further downwards.

The temperature of the patient is seldom raised more than a degree, the highest point reached being 100° F. In this particular case the boy was suffering from mumps at the same time. The moderate amount of pyrexia is disproportionate to the extent of the swelling and brawny condition at the orbital margin, and is a valuable means of diagnosis, especially in young children, where the tissues are easily distended, and where it is difficult to make a thorough examination without an anæsthetic. In one case, Jane C., orbital cellulitis was in the first instance suspected, until the temperature was found to be only 97° F.

Malaise to a moderate degree is experienced; it is more marked in the younger patients.

As the disease progresses the eye becomes completely closed, and the upper lid cannot be raised voluntarily. Eversion of the lid is extremely painful, but the gland can be exposed by gently lifting forwards the lid from the globe. It is often covered by the folds of œdematous ocular conjunctiva, which may also obscure the cornea. The skin of the lid becomes stretched and brawny, and it is at this stage that diagnosis is frequently wrong. The condition is taken to be one of abscess of the lid, or periostitis of the orbital margin. An incision through the skin for the relief of the former has failed on two occasions to evacuate pus, which later found an opening through the conjunctiva.

In the early acute stage it is impossible to say whether or not suppuration will take place. The probabilities, however, appear to be in favour of abscess formation, since 8 of the 10 cases here recorded suppurated.

Under an anæsthetic, the gland may be examined by inserting the forefinger into the upper fornix of the conjunctival sac, and palpating the tissues between it and the thumb placed externally. In this way fluctuation can sometimes be obtained. An examination from without to decide the presence of fluctuation is usually futile, owing to the want of resistance of the orbital tissues, and the deep position of the abscess. With the finger in the fornix, the limits of the swelling can be found laterally, but posteriorly it cannot be defined, showing that the palpebral portion of the gland is not alone affected.

Should resolution take place, the pain and swelling abate about the 3rd or 4th day; the lid slackens and can be raised. The conjunctival œdema disappears within 24 or 36 hours, and the malaise is followed by a speedy convalescence. The gland itself, however, although diminishing rapidly in size at first, as the acute symptoms disappear, does not resume its normal dimensions for 2 or 3 weeks.

Should suppuration take place, the symptoms continue, and are aggravated, whilst the physical signs become more marked. The conjunctival swelling is so great that the œdematous membrane may be extruded between the lids, and there retained, causing still greater swelling of the external portion. Even at the height of suppuration the temperature is not much raised.

At a varying period from the onset of the inflammation —from 1 day to a week afterwards—the patient feels the tension of the parts to be relieved. A thick purulent discharge issues from the conjunctival sac simultaneously, and within a few hours there is a marked improvement in the local condition.

The profuse discharge does not last more than 24 hours as a rule. Pus can be seen oozing from the opening in the

gland during this period, but later the discharge diminishes rapidly in amount, and soon becomes little more than catarrhal secretion from the conjunctiva.

Several writers have mentioned that cases of abscess of the lacrimal gland may point externally through the skin of the lid, leading to the formation of a fistula. Indeed, according to Nettleship's text book, "when an abscess forms it usually points to the skin, and should seldom be opened from the conjunctival surface," and Theobald says that "it may make its way to the surface through the integument of the lid, or may discharge into the superior conjunctival *cul-de-sac*." In none of these cases here recorded did the pus appear on the surface, and it is difficult to see why it should do so, since suppuration may proceed much more easily along the gland tissue, and shows no tendency to extend into the neighbouring tissues.

A probe, when introduced through the opening in the gland, passes deeply. The rough walls of the abscess cavity can be felt, but the wall of the orbit does not appear to be affected in any way, and is certainly not exposed.

Bock, in an exhaustive article on this disease, states that the accessory gland is not always involved, or, if involved, is not affected to a similar degree, whilst Nettleship, as before-mentioned, has met with cases of affection of the accessory portion alone.

Bock's observation cannot be substantiated from the above cases. In all of them, where an abscess formed, the two portions of the gland appeared to be matted together, the anterior part of the mass being distended downwards almost to the margin of the upper lid. In one instance of probable affection of the accessory gland due to extension of the inflammation of a suppurating Meibomian gland, which had discharged through the skin near the external canthus, the finger in the upper fornix could make out the posterior limit of the involved tissue. There was no evidence of affection of the deep gland, or of pus formation in the accessory portion. The fact that in several of those cases

operated upon, about a drachm of pus was evacuated, suggests that the greater part of the gland tissue was destroyed, the only healthy tissue being a thin shell forming the abscess wall. There was no reason to believe in any single case that the suppurative process was not wholly limited to the gland tissue.

Both during the height of the disease, and in the later stages, it was impossible to say whether or not the function of the gland was in abeyance. In most of the cases there was a little catarrhal discharge from the conjunctival sac, but there was certainly not an excessive amount of tear secretion. The secretion from the conjunctiva was sufficient for the purposes of lubrication, and, notwithstanding the heat of the parts, and probable desquamation of the conjunctival epithelium, dryness of the eye was not complained of at any stage.

The intensity of the inflammation, and the breaking down of gland tissue in some cases, would lead one to expect complete atrophy subsequently, but this was not observed in this series. The accessory portion can be exposed many months afterwards, and no difference can be detected between those of the two sides. Of course, the gland tissue must largely be replaced by fibrous tissue, and it may be the fibrous remains which are exposed to view on everting the lid, and the absence of any dry sensation of the eye due to abolition of tear secretion would not be surprising, since the lacrimal gland can be completely extirpated without any apparent want of moisture following its removal.

It is curious that the swelling of the main part of the gland was not observed to cause displacement of the globe, or to interfere in any way with the movements of the eye. Bock relates that one of his cases, and five of those recorded by Von Reuss, showed no displacement of the globe; in the others it was pushed downwards and inwards.

In one of the cases, William V., recorded above, proptosis was noted 2 days after the abscess had been opened. This was not observed before the operation; it was only

slight, and cannot be explained, since the swelling of the tissues had subsided to a considerable extent.

Even if mechanical displacement did not occur, it is difficult to see why Tenon's capsule should not become affected by the neighbouring inflammation. There was no sign of this complication. The eye could be rotated in all directions quite freely without causing any pain, and abduction appeared to be as easy as any other movement, showing the external rectus to be not involved.

When the height of the inflammation has been passed, the elevator of the upper lid soon resumes its full action, but for some weeks afterwards there is some fulness of the upper lid in its outer half, causing a convexity of the palpebral margin, which gives the patient an appearance of winking.

The gland itself may remain enlarged for a period of a month or 6 weeks afterwards.

Of the 10 cases observed, 6 occurred in males and 4 in females. Several cases other than those above described have come under notice during the past 2 years, but are not included in this series on account of their course not having been closely followed. In all of these, the sexes appeared to be equally affected. The age at which the disease may be expected shows wide limits. In Bock's investigations the earliest age was 8, and the latest 77 years. In the present series, there were 3 patients of the ages of 3, 4½ and 6 years respectively, whilst the oldest individual was 37.

That it is an affection of youth is indicated by the average age being 16, and acute inflammation is certainly more severe in the young patients.

The left side is much more frequently attacked than the right side, 9 of the 10 cases being on the left. No cause can be assigned for this marked difference.

Several writers have commented upon the association of dacryo-adenitis with swelling of the parotid gland, and

Bock, discussing the causation of the disease, suggests that some of these cases may be mumps of the lacrimal gland, the latter being only a modified salivary gland. He adds that the swelling of the parotid is especially noticeable in sub-acute and chronic cases.

Lawson states that a case of bilateral sub-acute inflammation of the lacrimal glands under his care seemed in its course to be very analogous to an attack of mumps, although there does not appear to have been any involvement of the salivary glands.

In only one instance of the present series was this interesting complication present. The affection of the salivary gland preceded the dacryo-adenitis by only 24 hours. The glands of the opposite side were normal throughout the course of the disease. Suppuration occurred in the lacrimal gland, but not in the salivary gland. According to Osler, suppuration is "an extremely rare complication in genuine idiopathic mumps." Yet in the only case occurring during an attack of mumps, an abscess formed, and was evacuated.

This circumstance makes it difficult to decide into which of the two groups before mentioned this case should go. Although associated with mumps, there was present neither of the characteristic features which seem to distinguish it as one of the first group, and the fact that the lacrimal gland suppurated whilst the parotid resolved in the usual manner, suggests that the lacrimal disease was merely coincident with the parotitis, a different form of infection having taken place.

In reviewing the cases under notice, one is forced to the conclusion that in every instance the disease was a purely local condition, as the symptoms disappeared rapidly as soon as the pent-up pus had free outlet.

With regard to the entrance of pyogenic or other organisms, attention may be drawn to the impetigo of the nostrils, and the blepharitis from which Jane C. and Leslie F. respectively suffered on admission to the hospital, and

to the severe phlyctenular conjunctivitis and pustular blepharitis which subsequently developed in Jack D. and Nellie W. In the latter two cases it might be argued that the conjunctiva was infected secondarily, but a sufficient time had elapsed in each instance for the whole inflammatory attack to have subsided, and both patients had been seen in a normal condition in the interval. There was no relapse of the dacryo-adenitis.

The lacrimal sac was invariably healthy in the series.

Bock lays stress upon the acute infectious diseases as precursors of the suppurative form. This cannot be substantiated from these cases, as there was no history of immediately preceding illness. In one child, of about 3 years, however, whose notes cannot be unfortunately traced, an attack of measles had occurred rather more than a fortnight previously, and the inflammation ended in the formation of an abscess.

The diagnosis of acute dacryo-adenitis presents little difficulty. When the inflammation is at its height, the closure of the palpebral aperture, the chemosis of the conjunctiva, and the great swelling of the tissues of the lid, have led to a diagnosis of orbital cellulitis. But, notwithstanding the tenderness of the parts, an examination of the upper lid soon demonstrates that the induration is confined to the outer part, the rest of the lid being swollen in a more or less passive manner. The fact that the lid cannot be everted at this stage without much difficulty and pain accounts for the prominence and congestion of the gland remaining unobserved, whilst the marked œdema of the conjunctiva, which may entirely obscure the cornea, give an impression that the globe is involved. The comparatively mild constitutional disturbance, moderate pyrexia, and absence of ocular symptoms, help to a correct conclusion as to the nature of the case. Examination of the upper fornix, under a local or general anæsthetic, according to the age of the patient, is conclusive.

Periostitis of the orbital margin has been suggested in

moderately severe cases. In such instances a sulcus could always be felt with the tip of the finger, between the point of greatest swelling and the rim of the orbit, and once, where tenderness was not a marked feature, the swollen gland could be moved freely against the bone.

The treatment of acute dacryo-adenitis is simple, and consists of hot fomentations applied frequently to the part. Theobald advocates "leeching, and the liberal application of an ointment of mercury and opium or belladonna, together with the administration of an energetic calomel cathartic, to be followed by free doses of quinine, salicylate of sodium, or pyrophosphate of sodium." This, it is hoped, would cut short the attack. If ineffectual "poultices or warm fomentations are to be applied, and, as soon as the presence of pus can be detected, it should be evacuated by a free incision, made either through the integument of the upper lid, or from the conjunctival *cul-de-sac*, as may seem to be indicated by the tendency of the abscess to point."

Concerning the treatment adopted in dealing with the cases recorded the latter part of this scheme was practically carried out, but no attempt was made to cause the condition to abort, since in the non-suppurative inflammations amelioration of the symptoms was obtained at once by the application of moist heat. Where pus is present, or, rather, where the severity of the symptoms and the extent of the swelling justify the assumption that an abscess exists (for fluctuation cannot always be obtained), an incision should be made, but never through the skin. The disadvantages of the latter method are the production of a scar, and the possible formation of a lacrimal fistula. But, as far as one can judge, it is never necessary, as the abscess tends naturally to discharge itself into the conjunctival sac.

The procedure adopted was to anæsthetise the patient if young, evert the lid if possible, and, by pressure at the orbital margin, force the gland to present. It was then incised with a Beers' knife, and, if pus did not appear, a pair of sinus forceps was introduced, and the aperture enlarged

In the youngest patients it was found necessary to slit the external canthus in order to get a good view of the structures behind the lid.

Fomentations were then applied, and recovery quickly followed. Whilst the discharge was profuse, boracic acid ointment was used to protect the cornea, and the conjunctival sac was frequently irrigated. Constitutional treatment was never needed.

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